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PRESIDENT'S ADDRESS

SOME CLINICAL OBSERVATIONS IN INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN.

J. A. Stucky, M.D.
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The attempt to discuss all the intracranial lesions of otitic origin would be unwise and impractical because of the time it would require and my lack of experience in dealing with many of them. Hence I have taken the liberty of changing the topic assigned me, so that I may present only those phases of these complications which I have met with in my own practice. The general discussion which is to follow the other papers on the program will consider probably all the forms of intracranial complications resulting from otitis media, and in this more practical way will the entire subject be brought before the Academy. The topic is too important and time too short to attempt to bring before you a résumé of the literature of the subject. We gain more from the recital of individual experiences than from lengthy dissertations and conclusions drawn from others. This is a great American Academy, where each gives the other his personal academic clinical experience learned at the bedside and operating table and often verified in the postmortem examination.

It is said that the father of otology, Professor Adam Politzer, in his introduction to his annual lectures on otology, takes up the temporal bone, and, holding it in full view of the class of students, slowly and forcibly tells them: "The temporal bone has four sides, the outside is bounded by life, from which there comes through the opening of the external auditory canal one form of our appreciation of what life means; on the other three sides this bone is bounded by death." There is much truth and pathos in this statement, especially when intracranial lesions are the result of a diseased condition of the temporal bone, but the dangers of years ago, when this grand old otologist first uttered these words, are being rapidly overcome by the advance of the medical and surgical science of to-day.

The only intracranial lesions of otitic origin I have met with are meningeal irritation (meningism), meningitis, extradural abscess and cerebral abscess involving the spheno-temporal bone, and the lessons learned from those cases lead me to the discussion of the topic, "Some Clinical Observations in Intracranial Complications of Otitic Origin."

The number of cases which I have observed and whose histories have been recorded is twenty-four, and each case was characterized by marked meningeal and cerebral symptoms, which operation revealed were of otitic origin or complication, and were not of that class so often found in doing the mastoid operation which had not previously given any symptoms of intracranial lesion. Of this number, seventeen were operated on, ten of which recovered. In seven others, all the symptoms were improved by the operation, for from one to five days; three cases were meningism as described by Chevalier Jackson, which were of otitic origin. In the seventeen cases operated on, one or several of the following conditions were found:

(a) Two cases of abscess of sphenotemporal lobe.

(b) Five cases of extradural abscess in middle fossa.

(c) Four cases of extradural abscess in the inferior fossa.

(d) Three cases of serous meningitis in which dark-colored fluid was found under considerable pressure between the bone and dura in the middle fossa.

(e) Seven cases in which the tegmen tympani and antri were destroyed and the dura thickened (pachymeningitis).

(f) Six cases in which the sinus was exposed; two were opened in removing the granulations covering them.

(g) Two cases in which the facial ridge was eroded, exposing the nerve, temporary palsy resulting.

(h) Five cases in which there were fistulous openings in the tegmen tympani.

(i) Six cases in which there was optic neuritis (in both the sphenotemporal abscess cases and in four cases of extradural abscess in the inferior fossa).

(j) Four cases were due to acute otitis media.

(k) Thirteen cases were due to a chronic otitis media.

In none of my cases was there a lateral sinus thrombosis or cerebellar abscess. Seven died without operation, and seven died who were operated on. Of the latter I was able to obtain autopsies in six, two of which showed tubercular meningitis, with the center of infection over the area of the tegmen tympani of a chronic suppurative otorrhea; three were diffuse purulent meningitis, one was diffused meningitis with suppuration of the Gasserian ganglion complicating double mastoiditis. Since operation on intracranial lesions has proven to be life-saving, the interest of the otologist and general practitioner has become more active in these patients. "In the ordinary neglected otorrhea swarms of micro-organisms are found, both saprophytic and pathogenic. The mere chronicity of an otorrhea without extension of serious disease is no guarantee to the individual of future immunity. Patients so affected are at any time liable to the rapid onset of dangerous sequelæ, which may arise from fresh inoculations of the discharge in the middle ear, or from fresh exposure of fresh area of the membrane brought about by progressive disintegration, and the implication of these fresh areas in the pathogenic process" (Macewen).

Chevalier Jackson says: "To look over the text-books and the periodical literature one would suppose that the only pyogenic extension of purulent otitis media was by way of the mastoid cells. Yet we all see cases of intracranial extension through the tympanic roof. Are we then justified in considering mastoid empyema the only indication for opening the mastoid? Are we not culpable when we fail to advise and urge early operation upon our patients with chronic otorrhea who are carrying about a foul discharge, offensive to their companions, rejected by insurance companies, and perhaps living in dread of an operation which they will likely come to sooner or later? The risks of operation are practically nil; the risks of postponement are great. To operate after an acute pyogenic invasion has affected the arachnoid, pia mater and cerebral fluids, while not a forlorn hope, is hazardous and the percentage of recovery reduced to more than one-half by the delay."

Symptoms of the several intracranial complications of otitic origin, in the initial stage, are similar, and it is impossible without submitting the patient to the danger of deeper and more extensive infection, to wait for focalizing or pathognomonic evidence. For this reason emphasis should be placed upon the danger especially of chronic suppuration of the middle ear extending to the cranial cavity and sinuses of the dura, and at the first evidence of meningeal irritation, as manifested by persistent headaches, vertigo, vomiting, somnolence or irritability, the radical operations should be performed, the dura exposed, examined, irrigated and drained through the tegmen tympani and antri. It matters not whether there exists symptoms of mastoid infection if the middle ear shows evidence of infection or there is a history of chronic suppuration having existed, I believe it best to advise immediate operation.

"The infection most frequently takes place through fistulous openings in the tegmen or the walls of the temporal bone toward the dura and sigmoid sinus. Infection less often occurs without any demonstrable lesions in the bone, by means of the numerous anastomoses between the blood and lymph vessels of the cranial cavity" (Politzer).

In some of my most pronounced cases of intracranial disease no pus was found at the operation and no evidence of active trouble existed in the tympanic cavity at the time, but the tegmen of the antrum, attic and tympanum were covered with granulations and the bone was soft and soggy and gave the sensation more of cutting through chalk than through bone.

I am convinced, as pointed out by Körner, that in the early stage of infection of the basal meninges inflammatory edema may produce the same symptoms as observed in extradural abscess. In three of my cases which recovered, in which the extradural abscess of the tegmen associated with compression of the overlying temporal lobes and the cerebral injection was disproportioned to the extradural lesion, while there was no recognizable abnormality of the intervening arachnoid, by mere contiguity without plastic exudate, the infection had passed through and red softening had begun, which in a few days more would have formed a sphenotemporal abscess. In such cases early operation, in which free drainage is given and the infected area thoroughly irrigated, gives promise of favorable results.

The five extreme cases which I had recover add increasing emphasis and hope to the plea for early operative interference in the class of cases under consideration, and I quite agree with Randall and others in the contention that these cases and also those of tuberculous meningitis ought to be operated on and often irrigated with warm normal saline solution. In only three cases have I used lumbar puncture, and I am not sure that appreciable benefit resulted. In cases of serous meningitis I shall continue to resort to it, but am convinced that drainage and irrigation through the middle fossa is the better treatment. I have washed away quantities of purulent exudate in some of my fatal cases and, though too late to avail more than temporary relief, I am sure the procedure was correct.

Cases I have met with have been few and of the more simple form of intracranial diseases of otitic origin, and some of them are open to question, but I am convinced that the suggestion of operating early is the safer to follow, and I have reason to believe that had I not waited so long in my first series of cases, and had my technic been better, the results would have been different.

"To operate on a patient who will certainly die is a discredit to otology and prevents or delays consent in other operable curable cases. To operate on a child whose symptoms are due not to the extension of its ear disease but to the toxemia of an acute infection is to make a grave mistake. To fail to operate on an operable curable mastoid or middle-ear disease which is producing grave symptoms is a fearful error" (Jackson). To all of which we agree, but in this age what excuse can there be for allowing a patient, except in very rare instances, to become hopeless before giving him the chance that only surgery can give? I believe many of the hopeless cases and fatal results are due to delay and halting between two opinions. In these cases everything is to be gained and nothing to be lost by the operation, and if the end is hastened a few hours and made easier by operation, both relatives and attendants have the consolation of the knowledge that all was done that could be. A forward step will be taken when our clientèle are taught this and also the wisdom of an autopsy whenever there is a reasonable request made. It is only in this way that the people and the profession will be made safer than they are now.

It must be remembered that there is such a difference in the contour and structure of the epitympanum and the mastoid as to justify the assertions that there is no bony structure in the human body which presents so many deviations from an arbitrary standard type as the normal epitympanum and mastoid process of the temporal bone. The same multiplicity of variations is found on the inner walls separating the mastoid from the cranial cavities. It is also true in intracranial complications of otitic origin; the definite symptoms of their existence unfortunately are either conspicuous by their absence or fallacious as to their significance, while the general condition of the patient may be such that the main localizing symptoms are incapable of demonstration. Especially is this true in cases of cerebral or cerebellar abscess. An exact diagnosis as to the location is often impossible and more than one operation may be needed before the desired results are attained. In epidural abscess there frequently are no general symptoms of meningitis, Nature walling off the area of meningeal inflammation from the cranial cavity and often thus effecting a spontaneous recovery.

In the cases of extradural abscess seen by me all but three were epitympanic; the others were in the posterior fossa and were the only ones giving evidence of threatening serious brain trouble before operation, these being of size and sufficient virulence to produce pressure and toxemia. In the former cases there was no destruction of the bony walls bordering on the dura, the infection having taken place through a fistulous perforation in the wall. Had the bony roof of the tympanic and antral cavities been destroyed by the pathological process, pus could not have collected between the dura and the bone.

Of course the more marked the symptoms of intracranial complication the more urgent is the indication for operation, but my own observations justify the conclusion in cases of otitic disease, whether acute or chronic, that if the threatened symptoms of meningeal irritation characterized by headache, photophobia, pain over the entire temporal bone, especially above and back of the ear; whether there be evidence of mastoid disease or brain pressure, with drooping eyelids, furred tongue and loss of appetite, slow pulse and sluggish pupils, the indications for immediate operation exist. Other brain symptoms, such as somnolence, dizziness, vomiting, optic neuritis and nystagmus may occur associated with an extradural abscess, yet the appearance of these symptoms is not to be relied upon for making a differential diagnosis.

Operative procedures need not subject the patient to greater dangers than already exist, and therefore can be undertaken before the advanced stages of meningitis have set in. As in threatened serious abdominal disease, appendicitis and peritonitis, immediate attempts are made to eliminate the primary focus of infection, so in the treatment of otitic meningitis our aim should be to eradicate the primary suppuration in the temporal bone. Only recently has otology received the full recognition it deserves, and the general practitioner who holds in his keeping the present and future health and hearing of his clientèle is just beginning to awaken to his responsibility—of warning them of the necessity of the most careful and scientific treatment in the nasal and accessory sinuses. When this branch of the medical profession is aroused we will have fewer cases of deafness and intracranial disease. It is his province to warn parents of the danger of delay in having their children with nasopharyngeal disease, especially adenoids or earache, promptly attended to. Upon his shoulders, too, rests the responsibility of early recognizing the first evidence of intracranial irritation. The class of cases under consideration is seen first by him, and when the otologist is called they have usually passed beyond the initial stage and the alarming symptoms of meningeal irritation or inflammation are present. It is often impossible to state with any assurance what the symptoms indicate, whether a serous, pachy- or leptomeningitis. The one fact that now confronts parents, friends and medical attendants is that the enemy is upon them, and no enemy of the human body strikes with greater force and horror than the mention of meningitis or brain disease. The question now is not so much what phase of intracranial disease we have to contend with, but what shall we do? We are assured of the fact that unless the condition that exists is speedily brought to an end the patient has everything to gain and nothing to lose by our most heroic efforts. If the well-known and accepted symptoms of the disease exist, and there is evidence of suppuration in the ear, or history of previous suppuration and symptoms of meningeal irritation, the two things to be done promptly and thoroughly are, first, to start vigorous elimination by purgatives, diuretics and diaphoretics; these failing to relieve in a few hours, second, to drain the middle fossa through the tegmen tympani and antri after doing a radical exenteration of the tympanic cavity and mastoid cells. We lose valuable time and handicap our profession by waiting for differential points in diagnosis. Undoubtedly intestinal toxemia begins early in all ear diseases, and I know of nothing that so completely interferes with peristalsis resulting in obstinate constipation as does pain in the ear. Here the vicious circle is started, in which we have first constipation followed by faulty and perverted intestinal metabolism, disordered innervation, the urine loaded with indican, with the result that marked evidence is shown to the toxic condition of the patient. This is no time or occasion for small doses frequently repeated or exploratory punctures; something radical is demanded from both a therapeutical and surgical standpoint. This is an occasion when in the very beginning we must hasten the results of our efforts, especially from the therapeutical standpoint. My rule is to start active purgation with one large dose of calomel, five grains for a child under 4 years of age and ten to twenty grains for those older. Give the full dose not only for its purgative but for its antiseptic and sedative effect, to be followed in six hours by one ounce of castor oil, which is to be followed two hours later by one ounce of sulphate of magnesia. If the results of these do not indicate that the alimentary canal is empty and clean, a colon flush is given. No nourishment is given but milk or broth every three or four hours, and not then if objected to by the patient. This treatment will assist materially in determining whether we are dealing with a meningeal irritation due to toxemia from the alimentary canal, or whether the chief source of the trouble is elsewhere. In either event it is the best preparation for any operative procedure that is to follow. If the meningeal symptoms are unmistakable I do not think it wise to wait for the effect of the drugs upon the eliminative organs, but give the calomel just before starting the anesthetic and hasten its action as soon afterward as possible. In several instances where the patient was unconscious and in convulsions the calomel was given after placing the patient on the operating table, the preparations for the operation being done during the administration of the anesthetic. In five cases where the symptoms of meningeal irritation and brain pressure were most marked and the cases seemed hopeless as far as any treatment was concerned, this procedure of free elimination, drainage and irrigation resulted in the recovery of three out of the five.

In none of my cases were the meningeal symptoms increased by the operation, and I am convinced that early operation is less dangerous than waiting for an exact diagnosis.

Macewen's record of nearly 80 per cent. of recoveries in cases operated on for intracranial lesions stands unparalleled. He, with Jansen, Herman, Heine, Ballance, Körner, Lane and other well-known Europeans, operates at the first evidence of the extension of the infection to the intracranial cavity.

I know of no branch of surgery in which adequate hospital equipment, where trained assistants who are familiar with the technic of the operator, counts for more than in surgery of the cranial cavity. It is here that the "team work" and "system" so prominently brought out by the Mayo brothers are of paramount value. It is here that the anesthetist, operator, assistant and nurses are to thoroughly understand and do his or her part without loss of time or friction. The minimum amount of anesthetic is to be used, and in certain parts of the operation practically none is to be given; the minimum amount of laceration and bruising of the soft parts, concussion and jarring done away with by substituting Kerrison's forceps and rongeurs for the chisel and mallet. The use of the hot saline solution of a temperature of 115° F. from a fountain syringe keeps the field of operation clean, controls hemorrhage and keeps the dura warm and moist, as advocated by Sir Victor Horsley; the careful sponging with gauze wrung out of hot sterile saline solution, avoids bruising the delicate structures. In a word, success largely depends upon the thoroughness with which the operation is done, with the minimum amount of anesthesia, traumatism and shock, and the most careful sustaining of the eliminative and recuperative powers of the patient. In these cases an excess of indican in the urine is more to be feared than the presence of small quantities of albumen or sugar, so far as the ultimate results are concerned, and more is accomplished in the first twenty-four hours by giving saline solution per rectum or hypodermoclysis than by giving milk, broth or any other form of nourishment.

In conclusion I would urge that the keynote of the otologist dealing with intracranial lesion be "operate early," and I believe that the otologist is the one to do the intracranial surgery resulting from infection from the ear.

Starr says: "It is an unfortunate fact that less attention has been paid to brain surgery in this country than is merited." Whether this statement is all true I am not prepared to say. If it is, it is a reproach to American surgeons, but not so great a one as waiting for external evidence of pus formation in the mastoid or alarming evidence of septic absorption or intracranial infection before operating. Of this, however, I am quite convinced, as stated at our last meeting, that in the domain under consideration "there is much timber, tall and uncut," much food for thought and labor, much anatomical and physiological work to do, and a still greater field for study in the pathological realm. Laurels and gratitude await the patient, persevering and courageous investigations of our members as to the diagnosis and treatment of intracranial complications of otitic origin.

A BRIEF CONSIDERATION OF THE PYOGENIC DISEASES OF THE BRAIN OF OTITIC ORIGIN.

(READ BY INVITATION.)

JAMES F. MCKERNON, M.D.
NEW YORK.

In considering the pyogenic lesions of the brain of otitic origin I can simply give you my own clinical experience in the cases which properly come under this heading. Should there appear in various parts of this paper what might at first seem suggestions foreign to this subject, I can only say that to me they play no small part as etiologic factors in its development.

No department of modern surgery to-day has surpassed the advance made in the treatment of the brain complications of otitic origin. This is due to the fact that more accurate observations have been made at the bedside, by a closer study of the symptoms presenting, by calling to our aid the laboratory worker, and by a careful study and inspection of autopsy specimens, and the knowledge thus gained has enabled us to make earlier diagnoses. and, by instituting proper surgical procedures, to save a greater number of lives.

When we realize that fully 37 per cent. of all cases of brain abscess are a direct result of an extension of an infective process from the middle ear, and of that number more than four-fifths are caused by a neglected middle-ear suppuration, it is not difficult to comprehend how important it is to treat this condition in order that the structures adjacent to the middle ear may not become involved. This is one of the special etiologic factors in the production of brain lesions of otitic origin. of which I wish to speak more fully.

When a patient consults his physician about an otorrhea of long standing the doctor is very apt to instruct him to syringe his ear with some kind of an antiseptic solution, two, three or four times a day, as the case may be, without ever having examined the canal and middle ear to discover to what cause the discharge may be due. Or, again, the patient, or the patient's parents if the patient be a child, are told that it is a good thing for the ear to discharge and that nothing must be done to prevent it, as it is Nature's method of ridding the system of an impurity, and that when the discharge ceases the ear will be cured. Unfortunately it all too frequently happens that when such a discharge does cease it is because the drainage from the middle ear has become obstructed, and the pus that was making its appearance at the meatus has been diverted into another channel and is already affecting the mastoid bone or one or more of the intracranial structures. It is the physician's first duty when consulted by these patients to determine whether or not a necrotic process is present in the middle ear as the cause of such discharge, and if upon examination dead bone be found the patient or his parents should be apprised of the risk he runs if this process is left to continue unchecked. It is much wiser if the patient be made to accept the responsibility of the future outcome of the case, provided operation is advised and refused, than that the physician be censured later for not having communicated the fact to him. It is not a difficult matter for the family physician to make these aural examinations when first consulted by these patients, and if this is done the future of many a patient will be placed on a comparatively safer plane than when it is omitted.

To demonstrate how an infective process can travel from the middle ear and affect other structures I show you a specimen in which a process has occurred, which beautifully illustrates the pathway of infection from the middle ear to the mastoid antrum by way of Nature's channel, the aditus ad antrum. From this central mastoid cell a further infection takes place through the roof, affecting the dura at this point and subsequently involving the cerebral substance adjacent to it, so that suppuration takes place, and we finally have a brain abscess formed which is connected indirectly with the middle ear from which the suppurative process originated.

In this section of the temporal bone which I here show you the infection has traveled a much shorter route, namely, through the roof of the tympanum and the dura lining the inner surface of this bone had become involved, and later an extension of the trouble was communicated to the brain substance in this region.

It is in adults that we usually find the infection in this locality, caused, no doubt, by the long-continued suppuration so closely adjacent in the middle ear cavity. The first route of infection described is the one usually occurring in children and young adults, the reason for this being that so many times in children the mastoid becomes involved in a suppurative process complicating either an acute otitis or an acute exacerbation of a chronic purulent condition, which is not recognized, or if so, not opened, thereby allowing the poisonous element to remain in the mastoid antrum, where it subsequently creates trouble by a process of extension through the bone and dura to the cerebral substance.

Frequently Nature, by her most beneficent action, prevents an extension of the infection to the brain substance itself by granulations forming on the bone surface of the dura, and causing hypertrophic changes in its structure, and walling off, for a time at least, a further inroad of the infection. Such a condition is called a pachymeningitis and explains the cause of the persistent localized headaches of which these patients constantly complain.

A collection of pus is very frequently found in contact with the dura in this region, and when present is referred to as epidural abscess. As an illustration of this attempt of Nature to protect the brain substance from injury I show you a section of a skull with a perforation through the inner table, and a markedly thickened dura that protected the cerebral substance which it covers, and the brain within was found, upon autopsy, to be uninvolved. Several years ago, and in fact until within the last decade, many of the cases of so-called meningitis owed their origin to just such an infective process as the one described.

Another form of brain disease which we frequently meet as a complication of either acute or chronic middle ear disease is an inflammation of the cerebral substance itself, and is called encephalitis. It usually occurs following an operation for the removal of an extensive necrosis, where a large area of dura has been exposed, as in an obliteration of the sigmoid sinus, and the favorite site is in the cerebellar tissue. This condition was formerly said to be caused by an infection at the time of operation, as the time of its appearance was usually some four or five days following the dural exposure. I do not believe this to be so, but think the cause of infection is carried through the veins from the dura to the cerebellar tissue within, as the poisonous substance is doubtless already in the thickened dura, and manipulation and irritation of this structure at the time of operation serves only to disseminate it to the brain substance beneath, causing an encephalitis with usually a fatal result.

Another prolific cause, in the past, for the development of an intracranial infection, or for the permanent loss of audition or both, was the practice, formerly so much in vogue, of doing a so-called Wilde incision in cases of subperiosteal abscess in children and expecting Nature to complete the cure. Many of these cases, when so treated, continued to discharge through the opening made until a permanent fistula was established, carrying constantly from its source (the infected mastoid process) the products of decay. If later this fistulous tract became obstructed the pus usually found its way through a channel of lesser resistance, namely, the inner plate of the temporal bone, which has become diseased through long and continuous contact with the suppurative process, thus establishing an intracranial infection months and even years after such an incision had been made. In the few instances where no permanent fistulous tract was left and the tissues healed, there was still left a discharging ear, which later became chronic in character and in this way jeopardized the future safety of the patient.

When we have a case before us which presents all the physical signs of a subperitoneal accumulation of pus it should always be evacuated under anesthesia. But we should not stop with the simple evacuation of the external abscess, we must proceed further and open the mastoid process, remove the disease already there, as the external manifestations of pus, in the vast majority of cases, is secondary to a primary mastoiditis. There are a few cases met with which are an exception to this rule. They are in very young children, and in these the pus burrows up from the middle ear external to the mastoid and finds its way beneath the periosteum, usually at the petro-squamous suture. But even if the latter condition does exist, it is always safer for the future of our patients to explore the mastoid and thus save the possibility of any subsequent invasion.

In a personal experience of 162 cases of subperiosteal abscess operated on, in children whose ages ranged from 5 weeks to 8 years, pus was found in the mastoid process in 158 of the cases. In the four children where pus was absent there was a large amount of granulation tissue and softened bone found, so that I believe it is very unwise to merely evacuate the subperiosteal accumulation and hope that Nature will come to our aid and complete a cure which should have been accomplished by the surgeon.

Another phase of what may properly be called otitic brain disease is a thrombus, complete or partial, of the whole, or any part of the sigmoid, or lateral sinus, or adjacent blood currents, and occurs as the result of either an acute or chronic suppuration of the middle ear, although it is more frequently associated as a complication of a chronic purulent condition. It does, however, complicate acute cases, and when it does the infection is usually of a virulent type.

The symptoms as recognized to-day are somewhat more complex than formerly, this complexity being due to the fact that there are classes of cases differing very materially, symptomatically speaking, from the type known as typical, so that at the present time we are enabled to classify the symptoms as belonging to a certain group of cases which, combined with our clinical experience, places at the disposal of the practical otologist a group of fairly definite symptoms from which to make a positive diagnosis of the disease.

In[1] reviewing the symptomatology of this disease I shall follow the lines of my personal experience, and do so under three heads: First, the symptomatology in the typical cases; second, in the atypical; and third, in those where the bulb and sinus are primarily involved, without macroscopic disease of the mastoid process. As the majority of cases coming under our observation are those secondary to a mastoid involvement, or developing after the mastoid operation has been done, there are a few definite symptoms present which are fairly constant and which enable us without delay to recognize this condition.

SYMPTOMATOLOGY.

Temperature.—The symptom which I consider by far the most important is that of temperature, and depends upon the amount of septic material entering the general circulation, which, if it be large in amount, is at once followed by a rise from the normal to 104, 105 or 106 degrees F., and many times higher, and this is quickly followed by a remission to near the normal point or below. There may be only one rise during a period of twenty-four hours, or several may take place, depending on the rapidity and quantity with which the poison is entering the general circulation. If the patient be kept under observation for several days these exacerbations of temperature become more frequent and the variations greater.

Chills.—They are present in only a portion of the cases seen, possibly 50 per cent., and I incline to believe that this is high. When present they usually precede the rise in temperature, and in some patients are followed by profuse sweating. Many of the cases coming under our observation exhibit no definite chill, they merely complain of a slight chilly sensation, and frequently this is overlooked unless the nurse is on her guard and watching for such a manifestation. In a postoperative mastoid case, with frequent variations of temperature, these slight chilly sensations are quite as important as though a decided rigor were present. In a communication before the American Otological Society in July, 1899, particular stress was laid upon this point, and during the intervening years at least one-third of all the cases observed has borne out the importance of watching for this but frequently spoken-of symptom.

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[1] Partial abstract from an article read before the Section on Otolaryngology of the Am. Med. Association, June, 1907.

Pulse.—In case of phlebitis, when there is a sudden high elevation of temperature, there is a corresponding rapidity of pulse rate, ranging from 120 to 150 per minute. In cases exhibiting a lower temperature range the pulse is complicated by a collection of pus in the brain and it is much slower. The respiratory changes are but slight but increase during the hyperpyrexia, at times becoming as rapid as 40 or 50 per minute. This is especially noticeable in the younger patients.

Pain.—In many cases of phlebitis coming under our observation the degree of pain is greater than that present when only an ordinary mastoiditis exists, or than that subsequent to a postoperative case. The pain is usually referred to the side of the head and to the occipital region, and is many times localized in the region of the torcular. In a few of the cases where the internal jugular vein becomes infected early there is decided pain in the neck along its course, and occasionally this pain will radiate down over the chest of the affected side. In many of the cases the pain present in the neck is caused, I believe, by an infected chain of lymphatic glands rather than by any obstruction in the vein.

Nausea and Vomiting.—These symptoms are usually present at some stage of the disease, in a moderate or pronounced degree. They usually complain of nausea first, particularly on being aroused or moving the head. This may be followed by slight vomiting, which later becomes more pronounced after the drinking of fluid. It may, however, occur at any time, independently of the taking of solids or fluids, and the amount is usually small.

Intraocular.—In only about one-third of the cases coming under my observation have eye symptoms been present. This may be due to the fact that many of the cases were seen and operated on early, and had they been left until a later period of the disease intraocular changes might have taken place. In a small number of the cases, when the phlebitis has extended to the cavernous sinus, there is pronounced swelling and puffiness of the eye and lid on the affected side.

Vertigo.—I have observed its presence only in those cases of long-standing purulent middle ear involvement or where the meninges were affected.

Cerebration.—This has invariably been normal, except in advanced cases. While some of the patients are drowsy and do not wish to be disturbed, yet they answer questions intelligently when aroused. If, however, the disease be allowed to progress this drowsiness increases and is followed by evidence of impaired cerebration.

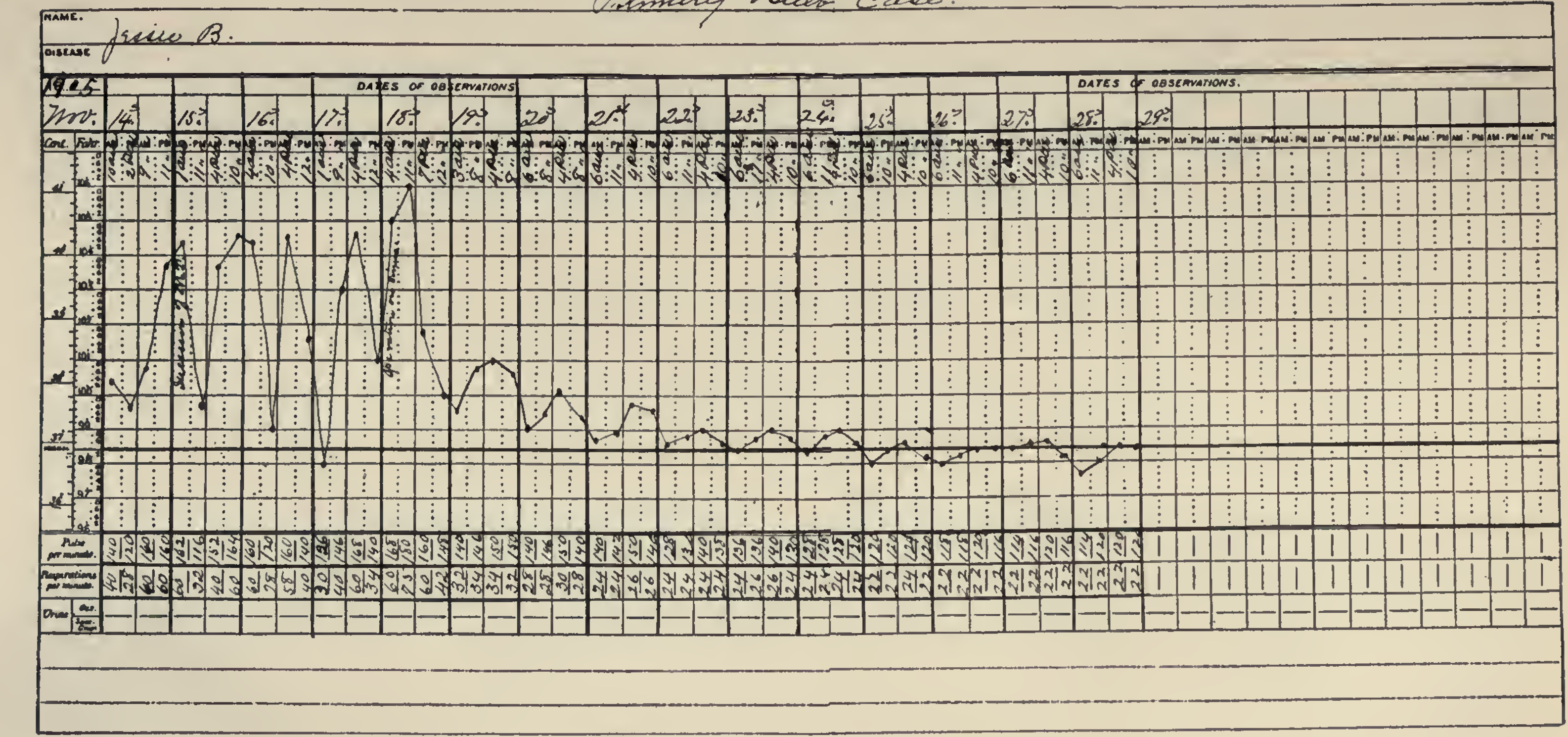
Physical Signs.—We occasionally find the presence of edema in the mastoid region, and edema over and around the exit of the emissary and occipital veins. In some of the well advanced cases, both in adults and children, there is a marked stiffness of the muscles of the neck on the affected side, showing a marked torticollis as it were. Combined with this there is a well-marked rigidity of the muscles present. A physical sign which the books tell us to look for in phlebitis is a hard, cord-like swelling in the neck. While unquestionably such a physical sign does not occur, in my experience it is very infrequent and only present in cases far advanced in the disease. I have never been able to demonstrate such a condition but once, though I have tried to do so repeatedly before operation.

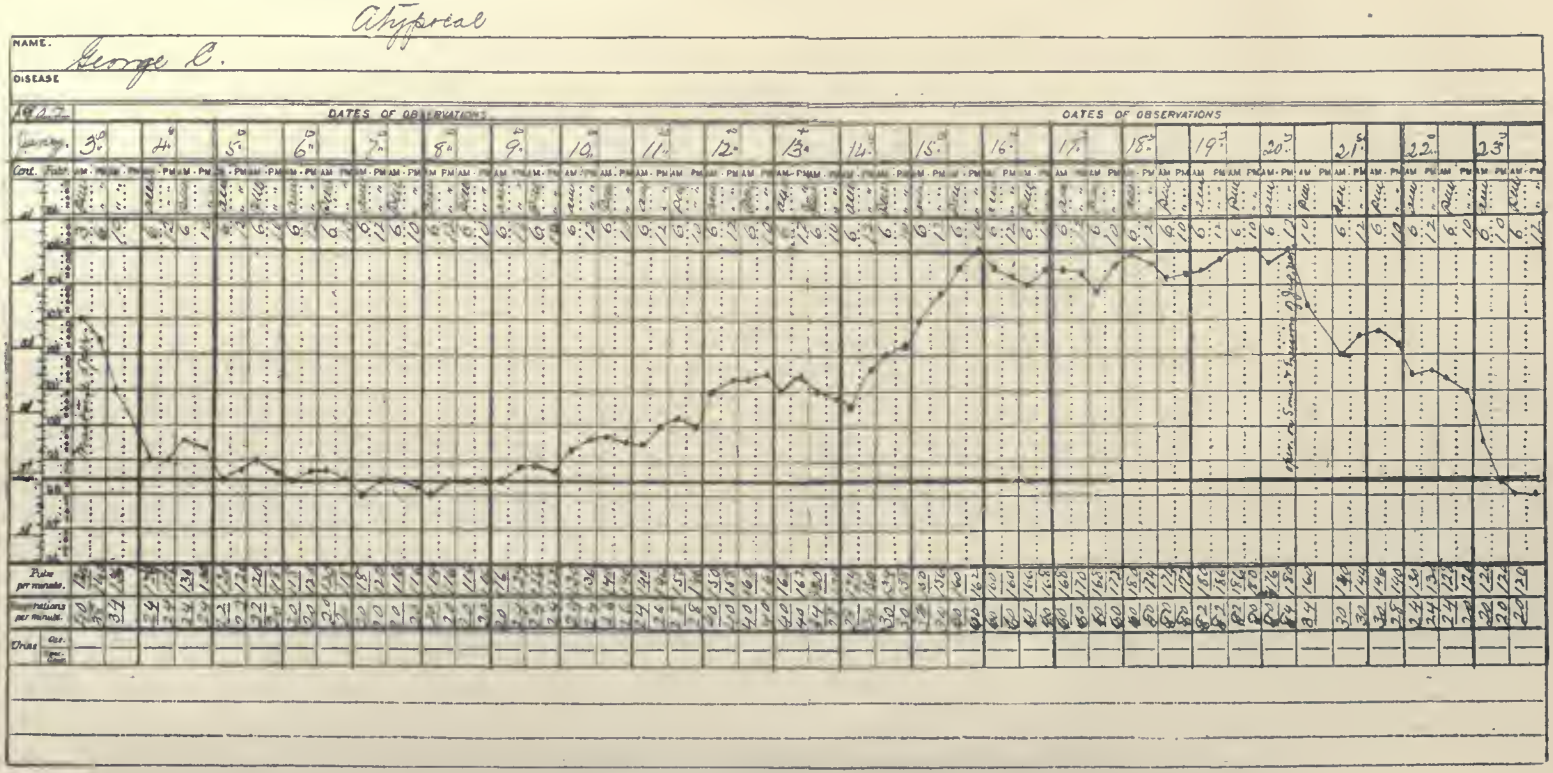
In cases well advanced in the disease metastatic deposits may occur in any joint of the body, either single or multiple, closely adjacent to, or remote from, the original focus of infection.

General Symptoms.—Among these may be mentioned loss of appetite, a dry and heavily furred tongue and a foul breath. The face is anxious, there is a pallid look, the skin is dry and later presents a yellowish tinge indicative of sepsis.

Atypical Cases.—These are cases invariably following a mastoid operation and usually do well for a few days after the mastoid has been opened. Then the patient becomes restless, irritable and disinclined to take food. The tongue, which before was clean and free from coating, becomes dry around the edges and presents a whitish, glazed appearance in the center. The pulse increases from 100 to 130 or 140 per minute, and there is a slow gradual rise in the temperature to 103, 104, 105 degrees F. or higher, remaining so for several days. In some of the cases it will not vary a degree in twenty-four to forty-eight hours, while in others there is a little variation, but there are no sudden drops and no sudden

Primary Bulb Case.




Atypical
NAME. George C.
DISEASE
DATES OF OBSERVATIONS
3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
AM PM
Pulse per minute.
Respirations per minute.
Urine

rises. The patients complain of headache and occasionally of nausea, but few reach the vomiting stage. They are unable to sleep for any considerable period of time, and if surgical measures are not instituted they later show all signs of pyemia. There is no chill or chilly sensations present; cerebration is clear, the only evidence of any mental disturbance being an occasional irritability of temper, and this is especially noticeable when they are first aroused. Later they become exceedingly drowsy, and the skin begins to take on a yellowish hue, and there is a profuse sweating.

Physical Signs.—A physical sign which I consider of the utmost importance in this class of cases is that when we inspect the mastoid wound, in the early stage of the disease, we find every portion of it apparently doing well and covered with granulations except at one point, and this point is the bone forming the sigmoid groove. We will find the bone in this region darker in color than when it was first exposed at the time of operation, and there will be no granulations found upon any part of it. In other words, we have a mastoid cavity showing all the evidence of healing except the bone over the sigmoid sinus. Should the infection progress farther and be allowed to go unchecked we will find in three or four days that the mastoid wound will present an altogether different picture. The parts that formerly were covered with healthy, firm granulations have now become pale and flabby, with a tendency to soften and break down, while the bone over the sinus still remains dark in color, perfectly dry and devoid of all reparative process. If during the mastoid operation the sinus was exposed we find at this stage the dura covering it presents a whitish or grayish appearance, devoid of luster, and at times covered with a plastic exudate, and if pressure be exerted upon this exposed area of the sinus it will be found easily compressible.

Bulb and Sinus Primarily Involved.[2]—In this third class of cases there is an acute purulent otitis present with the usual symptoms found accompanying this condition in an aggravated form, and the subsequent symptoms detailed are, I believe, the result of a direct infection from the tympanic cavity to the blood current closely adjacent, namely, through the floor of the tympanum to the jugular bulb. The explanation of the possibility of this is quite clear if we bear in mind the fact that in a certain percentage of the skulls examined we find an unusually high dome encroaching upon the middle ear cavity, or a dehiscence may exist in this region. Under such conditions as these it can readily be seen how an active purulent infection of the middle ear cavity can affect the blood current, without having first to travel its usual course through the venous and lymphatic structures of the mastoid bone: the pathway of infection is through the small communicating veins and lymphatics, or by a process of absorption directly through the thin wall of bone in this region.

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[2] Abstract of an article published in the Transactions of the American Otological Society, June, 1905.

The symptom in these cases of greatest importance is an unusually rapid rise in temperature (see chart) from 99 to 100 to 104, 105 or 106 degrees F. and as quite as sudden as the rise, a fall to 97, 98 or 99 or possibly 100 degress F. The temperature may remain low for several hours, and then quickly rise again to the points first mentioned, to be followed by a remission, and this may go on indefinitely until the end. During the exacerbations of temperature the pulse is rapid, ranging from 120 to 170 per minute. There is no chill present. The only evidence of one found in these patients is that if seen when the temperature begins to rise, they will be found to have cold hands and feet. They are exceedingly fretful and irritable, and later become drowsy. The eye signs are negative. If the temperature range is allowed to repeat itself for several days, the tongue becomes white and dry. During the temperature remissions these patients (and they are usually young children) feel remarkably well, will ask for food and ask to sit up and play with their toys. This is one of the phases of the disease which the parents and the uninitiated find hard to comprehend. They see such an apparent improvement in the little ones that they are misled as to the dangers existing, and often believe they are well on the road to recovery until the next rise of temperature occurs.

Take, for example, the symptoms as recognized to-day in a typical case of this disease. Briefly, they are the presence or a history of a purulent discharge from the middle ear, with a sudden rise of temperature, say from 99 or 100 to 105 or 106 degrees F., with a rapid remission, and this preceded by a chill, or a chilly sensation, occasional nausea and vomiting, and more or less irritability of temper with drowsiness. These are quite sufficient to notify the otologist that the patient's condition is not satisfactory. Should these symptoms be repeated in a few hours, there is evidence enough to warrant the exposure and exploration of the various portions of the sinus by the aurist.

In no branch of aural surgery of to-day have more brilliant successes been made than in the early recognition and operative treatment of this disease. Contrast this with the older methods of treatment, I shall not say recognition, for such a condition was not recognized a few years ago, and the cases were treated for almost every disease in the category until the meninges became affected, or they died from general sepsis or pyemia, and the cause in the majority of cases was not even dreamed of. To-day even many of these cases are treated for that much maligned disease, malaria, and later, when a general sepsis has occurred, if recognized, it is too late to save the patient's life by any operative procedure.

There is a phase of this disease that I wish to call particular attention to, because it is so misleading and unreliable. I refer to that stage of it which is present in a majority of the cases during the temperature remissions, namely: we have often been misled by the patient's apparent improvement at this stage, and during the earlier years of observation it was a most difficult and sometimes impossible matter to understand why, if the patient had such a serious condition present, he should apparently feel so well between the exacerbations of temperature. A close study of the cases as presented subsequently taught us that this could be explained by the fact that the rise in temperature is caused by some of the infective material in the sinus being carried into the internal jugular vein and disseminated through the body, thus causing a systemic poisoning. When this occurs we observe the chill, temperature, nausea, vomiting and marked drowsiness, with other symptoms of minor importance, but when the general system has eliminated this poison, or the major portion of it, we find our patients feeling brighter, more cheerful and inclined to be optimistic, as though they were far from any danger, when, in reality, the danger is just as imminent, if not more so, than before, for the symptoms enumerated recur again and again if the patient be left without surgical aid, until such a time that operative procedure, even though it be resorted to, is too late to save life.

Many of us can, I think, indulge in a retrospect and recall case after case of so-called idiopathic meningitis, and general pyemia that resulted fatally, when had the true cause been known the middle ear cavity was the starting point of it.

Diagnosis.—The first class of cases is easily recognized, our diagnosis being based upon the temperature changes and the presence of a chill or chilly sensation preceding the rise in temperature and following an operation on the mastoid. If most of the symptoms given in this class be present, it is, of course, comparatively easy to make a diagnosis, but if several are absent the diagnosis can be made from the temperature changes, with the chill or chilly sensations that are present. In those cases where the sinus is operated on at the same time that the mastoid operation is done, and we have no previous symptoms to guide us as to its involvement, we determine whether or not it is necessary to open the sinus at that time by the physical signs alone which are found presenting when the mastoid is opened. Briefly, these signs are, at times, the presence of a necrotic area of bone over and around the sinus, with usually an epidural collection of pus, commonly called a perisinus abscess. The dura covering the sinus wall is at times thicker than normal and darker in color, or it may present a lusterless appearance, darker at one point than another, and usually the lower end toward the bulb is of a whitish or gray color. At times there is a grayish plastic exudate adherent to the sinus wall, and the sinus looks flat and does not present a rounded appearance when divested of its bony covering, is easily compressible, and does not fill quickly when pressure is removed.

In the absence of previous symptoms referable to sinus involvement, the presence of an epidural or a perisinus abscess is not a positive indication for opening the sinus; it is only when the physical signs described above are present, with or without the epidural collection of pus; neither should the sinus be molested if the dura covering it be protected by granulation tissue, for this is an effort of Nature to combat the progress of the disease.

Bacteriological examination of the discharge from the ear is of value in certain cases, as when the bacillus of Friedländer is found the infection is prone to greater activity, travels more rapidly, and the sinus and vein are involved earlier than in many of the other forms of infection. Another valuable aid in diagnosis is the differential blood count, not so much for determining if a leucocytosis is present as to determine the polynuclear percentage. As, for example, if we find a differential count showing a polynuclear percentage of over 75, no matter what the leucocyte count be, whether great or small, we are almost certain to find an infective process which calls for surgical intervention. The leucocyte count is of value only in giving us information as to the patient's resistance to the disease.

An important point to be borne in mind in connection with the differential count is, that the polynuclear percentage is not as high when pus is present in bone cavities as when it is in the soft tissues of the body. As for example, take an ordinary pus infection of the mastoid bone free of complications; here the differential count will show, say, from 70 to 80 per cent. of polynuclear cells, and many will tell us that such a low percentage precludes the possibility of pus being present, which is not so. The explanation being that we have a bony cavity to deal with containing pus that is undergoing but little systemic absorption; but complicate such a bony cavity by an involvement of an adjacent blood current and there will be a rapid increase in the polynuclear cells from 85 to 95 per cent. and even higher.

In the second, or *atypical class,* occurring, as they do, some days after a primary operation on the mastoid, the diagnosis is made on the temperature changes and the patient's general condition.

Take, for example, a case of mastoiditis that has been operated on from, say, four to twelve days, the temperature during this interval following the usual course of a typical postoperative mastoid case; next day when we see the patient we find him a little restless, appetite, which before was good, variable, the tongue beginning to become coated, the pulse, which the day before ranged between 70 and 80, is now between 90 and 100, and the temperature that was normal has risen to 99.6 or 100.6 degrees F. or thereabouts. The next day the temperature will be from one to two degrees higher, with *no* marked remissions, the patient's general condition has changed somewhat, the restlessness is more pronounced, there is a distinct irritability, and he is disinclined to take food; the tongue has become dry around the edge with a thicker coating than the previous day, the pulse has increased in rapidity from 110 to 120 per minute, he complains of headache, not localized but general, and an occasional feeling of nausea, and is unable to sleep for any considerable period of time. When seen the following day the temperature has gradually risen to 104 degrees or higher, the pulse is now running from 120 to 140 per minute and shows a diminution in tension. The headache is more pronounced and the restlessness, sleeplessness and irritability are prominent features which can not fail of recognition. The white or grayish coating on the tongue has now changed, is brown and dry like leather and has a glazed center. If the disease is allowed to progress without efforts to combat it, we find within the next twenty-four hours the temperature has risen to 105 degrees F. or even higher, the pulse has become more rapid, 140 to 150 per minute, showing less volume and becoming weaker, and coincident with this is a refusal to partake of food. At this stage they become drowsy and exceedingly irritable when aroused, and wish to be let alone. As the disease progresses the temperature will practically remain stationary. with no marked remissions (see chart). The pulse gradually becomes weaker and we have the evidences of a general pyemia present.

A very valuable aid to us in diagnosis in this class of sinus disease is the physical sign spoken of earlier in this paper, namely, the absence of granulations over the inner table covering the thrombosed area, and its change in color from normal to dark. Another physical sign which aids us is, if the sinus was exposed at the time of the mastoid operation, we will find the dura covering it has undergone a marked pathological change, as it presents a grayish appearance, is lusterless and covered with a plastic exudate and the vessel itself looks flat.

In the third class of cases, namely, when the bulb and sinus are primarily involved, the diagnosis is made almost entirely from the rapid excursions of temperature closely following an acute purulent otitis. Bacteriological examination of the discharge from the ear is of value here as in other cases. The same is true of the differential blood count, and the utmost importance should be attached to it when the polynuclear proportion is found to be between 85 and 95 per cent., as I believe this is a positive evidence that the patient is suffering from an acute septic process. As a still further aid in diagnosis all other diseases should be ruled out by a process of elimination before the operation is decided upon.

Treatment.—While it is beyond the scope of such a paper as this to go into the details of treatment for such a condition, it may not be amiss to mention a few points in the technic. In dealing with this complication the treatment of the sinus will be spoken of first and, secondly, that of the vein when involved.

In this disease, as in many other surgical cases, each individual case is a law unto itself, and proceedings should be instituted as seem best to us at the time we are dealing with it. The first step is a complete and thorough exposure of the sinus, from a point above the bend or knee down to the region of the bulb, followed by a flushing of the operative field with a hot saline solution, and this by absolute alcohol to insure sterilization before opening the vessel. The treatment in the first and second class of cases does not materially differ. Too much bone should not be removed posteriorly, so as to uncover a large dural area over the cerebellum, as it is in that region that a hernia is liable to occur rather than at other points, and its occurrence is usually invited when considerable of the bony covering posterior to the sinus has been removed. After the sinus has been exposed, a small piece of iodoform gauze should be rolled up and placed over the upper end of the exposed sinus and pressed firmly under the edge of the bony groove, so as to cut off the circulation in the vessel should it be present; a similar piece of gauze should now be placed over the sinus in the region of the bulb and pressure exerted here, after which the dura forming the anterior sinus wall should be incised freely with a scalpel or scissors between these points and the contents of the sinus evacuated. The reason for abandoning the use of the aspirating needle as a diagnostic aid in these cases is because many times a thrombosed sinus might contain a small clot, while the remainder of the vessel would be filled with fluid blood; and unless the point of the needle engaged the clot, fluid blood would be drawn into the syringe, thus giving a wrong impression of the condition existing within, or a small parietal clot might be present and the needle would pierce this clot and then be passed further into the lumen of the vessel, drawing blood into the barrel of the syringe. Whereas, if we use a scalpel or scissors to open the sinus, a free incision being made, and the clot is a small one, it is washed out through the opening by the blood pressure from within. Should we be unable to obtain a flow of blood from the distal end of the sinus after the pressure has been removed in this region by taking the roll of gauze away, then we should make a further exposure in this region and proceed backward as far as the torcular if necessary, provided we do not secure a free return flow of blood before reaching it. For, if we do not obtain a blood current in the region sufficiently free to show that all obstruction has been removed, our patients do badly subsequently, either from an extension of the infective process to the blood channels or an encephalitis may develop one or two weeks later, caused by an extension from the infection we have overlooked in this region.

When a thrombus is removed and the blood flow established from the proximal end, hemorrhage should be allowed to continue for a few seconds, so as to remove any small clot or septic material that may be present further back in the vessel. This hemorrhage is then easily controlled by packing a small piece of folded gauze directly against the opening in the vessel.

The lower portion of the sinus is proceeded with in the same manner, and, after the removal of the clot and whatever pus and broken-down material may be present, an attempt should be made to restore the circulation at the bulb. In about half the number of uncomplicated cases coming under our observation, this can be done quite easily; but in others it becomes impossible, without dangerous manipulation; by this I mean, if very much time or effort be consumed by our attempt to restore the return circulation at the bulb, we run the chance of dislodging some foci of septic material from the bulb and having it thrown at once into the general circulation, thus increasing our systemic poisoning.

In the cases where the sinus does not contain pus, and the symptoms presenting show no evidence of a jugular involvement, it is better to cease our manipulations even though a return current of blood be not established; cleanse the operative field and pack the sinus firmly at the bulb with gauze rather than proceed any further, as experience teaches us that a large number of these cases recover without resort to further surgical procedure. This is especially true of the cases that are operated on early, before marked systemic symptoms have developed and before the coats of the vein below have become infected with the micro-organisms of the disease. In all these cases encountered, whether of the septic or non-septic variety, we should thoroughly expose the lower end of the sinus at the bulb, as here quite frequently is found the clot, and not above, owing to its close proximity to the floor of the tympanic cavity; this is especially true when we have a large jugular bulb to deal with. Care should be taken to excise all sloughing edges of dura, for if left untouched after the sinus has been incised they will retard the healing process. When pressure has been removed from the bulb portion of the sinus, we at times encounter a free return flow of blood, but whether the source of such a hemorrhage be from the internal jugular vein or from the inferior petrosal sinus we are unable to say, for it is as likely to come from the sinus as from the vein. But, when such a free return flow is present, and the sinus above contained, upon opening it, a parietal, or a firm and non-disintegrated clot, we usually end the operation here and await further developments. If, on the other hand, we find, upon exploring the sinus, pus or a disintegrated clot present, or both, we should desist from any further manipulations above, protect our already exposed field of operation, and proceed at once to expose, ligate and resect the internal jugular vein from a point below at the clavicle, to its exit from the skull. Should any of its tributaries be found involved, they should also be ligated and resected well beyond any point of macroscopic involvement. All diseased glands exposed during the resection of the vein should be removed. After this has been accomplished the contents of the sinus should be evacuated in the manner spoken of earlier in this paper. When such a procedure as this is carried out, it usually prevents the possibility of any further infection being thrown into the general circulation through the medium of this vessel. To be sure a still further infection may take place even after the vein has been resected through the medium of the anterior and posterior condyloid veins or the occipital and petrosal sinuses.

In many cases where the vein has been removed primarily, before the evacuation of the sinus contents from above, we find that the return of blood from the region of the bulb is quite as free as we would expect to find it if we had the internal jugular vein carrying on its usual function, the blood coming, no doubt, from the inferior petrosal sinus, and this is a valuable point to be remembered, for many operators will tell us that when we succeed in obtaining free hemorrhage from this end of the sinus it shows that the vein below is uncomplicated and is performing the function for which it was created, that of transmitting an unobstructed current of blood back to the heart, when, in reality, there is not a drop of blood present which has its source from the vein below. I have seen this demonstrated several times when the internal jugular vein was ligated and resected as a preliminary to exploring the sinus above. Occasionally after the vein has been removed we are unable to obtain a return flow from the bulb, which means that the inferior petrosal sinus is involved, and when such a condition confronts us (which fortunately is not frequent) we should, provided the patient's general condition will allow it, expose this sinus and remove the obstruction, as otherwise we are unmindful of the best interests of our patient by allowing an infective process to remain, which may at any time cause trouble. Should the superior petrosal sinus be found involved it should be exposed and treated in a like manner.

When the internal jugular vein is removed, the treatment of the neck wound is optional, either by the open method, with packing, and later drawn together by straps, or suturing at the time of the operation and inserting a large cigarette drain. I prefer the latter method with wet saline dressings, changed every twenty-four hours, and each time the dressing is reapplied, a small portion of the drain is removed, that at the end of the fourth or fifth dressing, the drain has been entirely removed, and primary union accomplished in the majority of cases.

In the primary bulb and sinus cases (the patients being nearly all young children), it would seem that the treatment should be the same as in the other cases, but here we must remember that these cases are caused by a primary infection of the blood current and when operated upon early the percentage of recoveries is in favor of removing the clot without ligating the vein, for these young patients do not bear prolonged operations well, and it is a matter of greater difficulty to ligate and resect the internal jugular vein in a young child than it is in an adult, for we have to deal with a neck that is short and chubby, and usually a number of enlarged glands are encountered, which prolong the operation, many times beyond the safety point. If subsequently ligation becomes necessary, it can be done rapidly and not subject the young children to the added risk of a prolonged operation.

It must be borne in mind that in this class of cases, when free hemorrhage is obtained from below, we can never be sure that all of the infective material has been removed, for it is impossible to pass a curette from above into the bulb, and in all probability a larger number of our cases operated upon without ligation of the vein have some infective material left in the region, but not in such quantity that the system can not care for it by a process of elimination. There is a type of cases in young children to which strong exception must be taken to the conservative method of treatment. I refer to those cases when, upon opening the sinus, pus or a broken-down clot may be found; when such a condition as this confronts us, whether there be free hemorrhage at the bulb or not, I believe it is imperative for the safety of our patient that the vein be removed at once and not wait for a train of symptoms that are too well known and unfortunately usually fatal to the patient.

There are many postoperative mastoid cases that fail to progress favorably, showing many or all of the so-called positive signs of sinus infection, and yet when these cases are operated on subsequently and the sinus explored no clot can be demonstrated, yet, from the moment the sinus is opened and a free hemorrhage takes place, the patient begins to improve and an ultimate recovery follows.

What is the explanation of this? Simply that we have a small parietal or central thrombus to deal with, and when the sinus wall was freely incised there was a sudden gush of blood outward, which carried the clot with it, and many times careful search will reveal such a thrombus adherent to the towels surrounding the operative field or to the gauze that has been used in sponging. In our septic cases when after operation the patient does not progress as favorably as we think he should, the temperature remaining high, there being only a partial subsidence of the general symptoms, and where we are doubtful whether any further operative procedure is demanded, it is wise to wait for a time before any other radical measures are taken, for we must remember the fact that we are dealing with a septic case, which took several days to develop, that there was a gradual absorption into the system of a virulent poison, and the postoperative history of such a case would show the slow elimination of the poisonous element absorbed, even though the source from which the infection emanated had been removed. Before leaving this important subject, I can not too strongly urge the necessity for an early operative procedure; we should not procrastinate until the infective material so thoroughly permeates the system that all possible chance to save the patient's life is gone, and the most favorable time for these patients to be operated on is during the temperature remissions, for, if done then, they bear the shock of the operation much better and resect more favorably than if operated on during exacerbations of the temperature. This point has been well demonstrated in prolonged operations, as in the resection of the vein and the removal of the infected glands following the mastoid operation.

One of the most frequent of the pyogenic infections arising from middle ear suppuration is pachymeningitis, and is a condition very frequently found complicating mastoiditis; when present the one prominent symptom always complained of by the patients is persistent and localized pain over the site of the pus accumulation. This, with marked tenderness upon pressure over the bone, may constitute the only prominent sign that is present.

The treatment of this condition is a free exposure of the dura, and the removal of all necrotic bone adjacent to it, but care should be taken not to separate the adhesions between the skull and dura, which has been Nature's effort to localize the process.

LEPTOMENINGITIS.

In former years when a leptomeningitis followed a middle ear suppuration it was invariably fatal because the infected area was seldom, if ever, exposed, cleansed and drained; or, if such a procedure was carried out, it was only after the patient's condition had become so serious that it would have been wiser not to have operated, the vitality being at such a low ebb that death followed quickly. It was left to the brilliant surgery of Macewen to early recognize and expose these infected areas and institute proper surgical methods for their relief and cure, for to-day a goodly number of these cases are saved by the treatment advised and first practiced by this eminent surgeon. Time does not permit of even a brief abstract of the symptomatology and diagnosis of this most important intracranial lesion. One point, however, may be mentioned that is an aid to us now in the diagnosis as well as treatment; I refer to lumbar puncture, for by an examination of the spinal fluid we are now able to make an earlier diagnosis as well as relieve some of the most distressing symptoms by drawing off a considerable quantity of cerebrospinal fluid, thus relieving pressure and in some instances in the severe cases prolonging life.

BRAIN ABSCESS.

This complication of purulent otitis is often very difficult of recognition because of the complexity of symptoms that may be present, a part of which may be due to other intracranial conditions than that of the abscess, and for this reason a positive diagnosis is impossible in many of the cases. In cerebral abscess the symptoms presented in those cases observed by the writer are briefly as follows:

Symptoms.—The presence or history of a purulent discharge from the ear of the affected side, persistent and localized head pain, tenderness on percussion, fretfulness, followed later by irritability, moaning, crying out in their sleep, and in many gritting of teeth, insomnia, nausea with vomiting, drowsiness, with intolerance of light, aphasia, depending on the location of the pus collection. Vertigo well marked in some cases. The nerve manifestations in the majority of cases have been of but little help in arriving at a diagnosis. Intraocular changes when present are valuable, but by no means positive aids. The pulse is slow, from 48 to 65 in cases uncomplicated by other intracranial affections; when complicated it is much higher and very misleading. The temperature in an uncomplicated case is low, ranging from subnormal to a little over a hundred, but in so many cases of the acute type the lesion is coincident with one or more of the complications, and here the temperature is much higher and again misleading.

Diagnosis.—The diagnosis is usually made on a majority of the symptoms enumerated; the differential blood count aids us very materially. In the abscess of acute origin before a limiting membrane has had sufficient time to become organized, there will be a greater amount of absorption and with this a corresponding higher polynuclear percentage, ranging from 85 per cent. to 96 per cent.; if the abscess is of the chronic variety from an old purulent condition of the middle ear, and of itself long standing, the polynuclear percentage will be considerably lower, owing to the fact that there is but little systemic absorption occurring, because the surrounding brain tissue is protected by a more or less dense and limiting membrane.

Another aid which may be of more value to us in the future than now is a radiograph of the cranial contents. In one case it has been of help to the writer. It has, however, been used so seldom that as yet no definite value can be attributed to it. It is to be hoped, however, that in the near future an improved technic may increase its value to us in this line of investigation.

CEREBELLAR ABSCESS.

A collection of pus in this region may remain unrecognized and give but little trouble for years; especially is this true if the accumulation be small, with well-formed walls, but if situated deeply the lack of coordination on the part of the patient makes us suspect at once that we have a lesion of the posterior fossa with which to deal. An abscess in this region of recent origin may present many of the symptoms as when located in other regions of the brain.

Treatment.—In searching for a collection of pus in the brain, irrespective of its location, a few points in the technic may be mentioned. It is well to bear in mind when we are searching the various regions of the brain for pus that the skull should be entered at a point corresponding as nearly as possible to the floor of the lobe we are about to explore, in order that free drainage may be secured. There should be sufficient exposure of the dura to enable the operator to thoroughly explore the region in all directions, for many times the otologist is at fault here, because he makes too small an opening in the skull and produces more injury to the dura and cerebral substance by so doing in his efforts of exploration than if the field of exposure were larger. When the dura has been exposed it is usually incised in its horizontal plane, care being taken to avoid severing any large blood vessels which supply it. After it has been incised, silk sutures should be passed through the flaps, as in this way they can replace the use of retractors, and draw the edges apart, and do not injure the dura; and should the brain exploration prove negative they can then be used to close the opening that has been made before proceeding to expose other portions of the dura. There are several methods of exploring the brain, and all have their advocates; my own experience has led me to rely upon the use of a long narrow-bladed scalpel, and if pus be encountered, to withdraw the knife and introduce into the opening made by it a pair of forceps with the blades closed and then enlarge the opening already made by separating the handles; this will make an opening sufficiently large to admit of the drainage of any cavity that may be found. The use of the finger to enlarge the opening made by the knife in the cerebral tissue is, I believe, fraught with danger to the surrounding tissues on account of the pressure it exerts. The cavity may be cleaned by one of two methods, depending on whether we are dealing with a pus accumulation of a recent or remote origin. If of recent origin without the presence of organized tissue to wall it off, then the least manipulation the better, and it can be cleansed by very gentle mopping with narrow strips of sterile gauze and drained by inserting a small wick of gauze down to the bottom of the cavity and allowing the brain substance to collapse around it, for it is a well-demonstrated clinical fact that brain abscesses of the acute type discharge but little after evacuation, and consequently do not need to be packed. If, however, we are dealing with an abscess of long standing, there will be, aside from the pus, considerable broken-down and disintegrated brain tissue which can best be removed by gentle irritation with a warm saline solution, after which the cavity can be mopped out as above with sterile gauze; drainage can be accomplished by passing a large wick down to the bottom of the cavity. This wick can be moistened and rolled in powder composed of equal parts of iodoform and boric acid, after the manner formerly advised by Macewen. But in addition to placing a wick in such an abscess cavity, the whole interior of it should be packed quite firmly with gauze to promote repair, for with its limiting membrane or connective tissue wall it does not collapse around the wick, as does the acute type of abscess. A very serviceable drain in use is also that of decalcified turkey bone.

Before draining these abscess cavities the utmost diligence should be exercised in searching for multiple abscesses which occur frequently, and are many times overlooked and found only at subsequent operations or upon autopsy.

There are few intracranial lesions of the pyemic type, otitic in origin, that I have not mentioned, such as a thrombus of the cavernous sinus, etc., but I have omitted them purposely, as they occur very infrequently.

In order that fewer pyogenic diseases that have been considered may occur in the future, it is our duty as otologists to impart to the undergraduates of medicine in our colleges the gravity of a running ear, so that when they are later confronted with this condition appropriate means for its relief can be at once instituted.

There is a subject of the utmost importance that deeply interests us as otologists to-day, and, though it is one that by no means can properly be classified under the heading of this paper, it is of such vital importance to both the aurist and the patient that I am going to crave your indulgence for a few moments longer to consider this important topic. I refer to the indications for the performance of the so-called radical, or Stacke, operation.

During the past five years there have been so many radical or Stacke operations reported done for the cure of chronic otorrhea that one is led to pause a moment and ask himself whether all were necessary or if some of the many cases reported could not have been cured by other and simpler means? I would in no sense be understood as belittling this operation, for I know of no other that offers such safety to the patient as this does when the indications are distinct for its performance. But from observation and reports I am inclined to believe that it is frequently done when little or no necrosis exists and before other means of a simpler and more conservative nature have been tried to cure the discharge.

What, then, are the indications for doing this operation? First, the presence of dead bone in the tympanic cavity, with or without the presence of cholesteatomatous masses; second, a train of symptoms occurring as the result of this necrotic area, which are, briefly, headache, localized or general, on the affected side, vertigo of the intermittent type, nausea and vomiting at times, intermittent pain, to be replaced by a dull, heavy, throbbing pain when drainage is obstructed; unsteadiness of gait in advanced cases, and an intermittent or constant purulent discharge from the middle ear, the odor of which is foul, and an indication of necrotic bone. Combined with these, in a certain number of cases, particularly when the process is of long standing, is a train of mental symptoms which in many of the cases produces a mild form of melancholia, caused by the patient's dwelling continuously upon the local process. Then, again, in a number of cases, there is a persistent and most distressing tinnitus, and frequently this has so upset the patient's mental equilibrium that all other symptoms are of minor importance as compared to this most distressing one.

When we have the history of a long-standing discharge from the middle ear, and upon examination we find dead bone present, with several of the above symptoms prominent, then I believe we have distinct and positive reasons for advising the performance of this operation. On the other hand, there are many cases in which the patient is advised to have this operation done simply because for a few weeks or months there has been a discharge from the middle ear, scanty in character, and when upon physical examination no dead bone can be demonstrated by the probe or other means brought to aid the examiner. In some of these cases the probe does encounter exposed bone where the mucous membrane covering it has been destroyed in the suppurative process, but an area of exposed bone should not be mistaken for dead bone. Again, take a case giving the history of an intermittent discharge from the ear for several months, with occasional attacks of pain and the audition but slightly impaired. When, upon examination, we find a drum membrane markedly thickened and swollen, with a small perforation situated some distance higher than the level of the tympanic floor, and the canal containing more or less foul-smelling secretion, because it has been allowed to accumulate there and decompose, a diagnosis of intratympanic caries is immediately made, or jumped at, from the character of the discharge, and an operation advised as the only means of a cure.

In cases like this I believe it is extremely unwise to do a radical operation on the middle ear cavity until all persistent and conservative treatment has failed to bring about resolution in these structures, and in many of the cases of a type similar to that described, cleaning and free incision in the thickened and swollen drum membrane, so as to freely drain the cavity behind it, and this followed by the simplest of measures, that of keeping the canal sterile, and stimulating the part as needed, will cure a large percentage of these cases, and at the same time preserve for the patient an audition that will be serviceable for the remainder of his life, and this, too, without inconvenience or loss of time. Whereas, if a Stacke operation were done on such a case, the hearing for all practical purposes, namely, conversation, would be a thing of the past, to say nothing of the inconvenience, suffering and loss of time to the patient.

Another class of cases frequently coming to the operating table for the Stacke operation is that of children giving the history of a discharge from the middle ear for several months as a sequelæ of grippe, measles, scarlet fever and other intercurrent diseases. In some of these cases, persistent conservative treatment has failed to bring about a cure. The adenoids and enlarged tonsils have been removed, hoping thereby to lessen the mechanical irritation in the nasopharynx so that resolution will be established, but without avail. If instead of the Stacke operation being done on these children, the mastoid were opened, the middle ear drained posteriorly, and at the same time a free incision were made in the drum membrane, the suppurative process would speedily cease, and, what is of the utmost importance for the future of these young patients, their hearing would be preserved, if not entirely, nearly so in the majority of cases. While if the so-called radical operation had been done on these patients, the hearing for all time would be very much diminished, if not entirely lost at the end of a year or two after such an operation.

During the last two years the writer has seen forty-four cases of the above-described type in children operated on by the posterior method, and all discharge cured except in two cases. The audition in thirty-eight of the cases was as perfect as the opposite side; of the remaining six cases, the audition was improved in four and two had to be operated on again, the Stacke operation being done at the second sitting.

In a certain number of cases of the adult type, where the only prominent symptom is that of otorrhea, scanty or intermittent, the duration of which is a few months or a year, and a section of the drum membrane is found wanting, it would seem wiser at first to perform ossiculectomy and curettage of the middle ear, hoping thereby to cure the condition by promoting drainage, rather than to at first do the radical operation, which, however, should be done later, provided the simpler measures instituted be not successful. The question naturally arises here, "How long should one be content with conservative treatment of a running ear before abandoning it and advising the patient to submit to the radical operation?" I believe it is impossible to lay down hard and fast rules as to the length of time a discharge may be allowed to continue without resorting to surgical procedures, as many things have to be taken into consideration: the duration of the discharge, its character, the previous treatment the patient has been subjected to, and, above all, his general condition, for this may be so depleted and below par that it is well-nigh impossible for a local condition to resolve while the patient's general system is so far below the normal health standard. Generally speaking, however, if two to four months' conservative treatment does not bring about a favorable result, then operative measures should be advised. This is, of course, if no evidence of a serious extension presents itself during this period. Should it do so, then a prompt interference is at once demanded.

When talking to patients about the advisability of having such an operation performed, the question arises as to how much or what should be said relative to audition, whether it will be good, or better than before operation, or, if it should be diminished, to what exent? The writer has seen a number of cases in both hospital and private practice where, after operation, the patient has volunteered the statement that had he known his hearing would be so poor as it was then he would never have had the operation done. Such a statement as this should have but little weight when we compare the diminished audition, with the possibility or probability of an intracranial condition occurring, in cases where advanced necrosis was present. It should, however, be judiciously weighed, in cases where the discharge has been of short duration, as many times these patients are prevented from earning their living at what was their former avocation after having submitted to the operation. So that I believe it to be our duty not to omit telling these patients when they come to us for advice that their audition may be permanently impaired by the operation.

There are a number of cases of this type operated on where brilliant results are reported from the standpoint of improved hearing immediately following the operation, we all know. But how about the hearing a year or two later, after a substantial mass of scar tissue has formed in the tympanic cavity? It should not be so much the immediate as the future result that we should endeavor to obtain for these patients.

Another point that these patients who submit to the radical operation should be enlightened upon is the possibility of an injury to the facial nerve. While none of us who do this operation expect this accident to take place, still there is always a possibility of its occurring, no matter how experienced or skilful the operator. Should such an accident occur, without our patient having previously been told of its possibility, it places the surgeon in a very unpleasant position, and for this reason, if for no other, it would be wise to acquaint a patient who contemplates having this operation done of the possibility of an accident occurring.

After a Stacke operation has been done the question in the mind of the operator is what to do to bring about a speedy dermatization of the tympanic cavity, and it is the belief of the writer that the primary skin graft seldom, if ever, hastens or lessens the duration of this process. Used secondarily, or several days after the operation has been done, it is, I believe, a distinct aid in the hastening of the healing process in a few selected cases. In the majority of cases where it is used either primarily or secondarily I believe it does but little good, as it sloughs from the surface on which it has been placed, or is torn away, or displaced during the earlier dressings of the case.

This conclusion is based upon a faithful and conscientious trial of it, both as primary and secondary graft. Without its use I believe the majority of cases operated on dermatize quite as rapidly, if not more so, than where it has been used. In the experience of the writer, the best means of effecting a rapid cure for the condition operated on is not the placing of a graft in the tympanic cavity and adjacent to it, but the complete removal of every vestige of the disease, and if this be done a cure will follow in nearly every case, independent of all other aid.

INTRACRANIAL LESIONS OF OTITIC ORIGIN.

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The repetition of an established fact may be considered by some as a waste of valuable time in a society of this character; however, the perception of new ideas and the application of old truths usually follow such a résumé, and, with this hope in mind, I shall endeavor to briefly review the past history of intracranial lesions of otitic origin without any effort to analyze each separate lesion or burden the society with the long scientific treatise necessary for a complete illumination of this subject up to date.

In a review of over three thousand references to disease of the ear and its sequelæ, now in the office of the Surgeon-General of the U. S. Army, I was much impressed with the above subject both in its historical and its surgical aspect. It is interesting to know that many of the contributors were pioneers in the science of otology, and have long since passed away.

Among the illustrious names is that of Zaufal, of Prague, who in 1880[1] was the first aurist to remove a blood clot from an infected lateral sinus and taught us the efficiency of ligation of the jugular vein for the prevention of metastatic infiltration.

According to Politzer,[2] Lane, of London, nine years later, was the first to report a cure of a case of sinus thrombosis by operation. Since that time, and especially in the last five years, a growing number of successful operations have been reported. However, this morbid process was recognized many years before, as shown by contributions from Weil in 1859, Sexton in 1865, and others.

In a consideration of cerebral affections due to otitic inflammation, which may often precede or follow any operation on the ear, the avenue of extension is of importance and may, by metastasis or direct extension by continuity of tissue, be through the roof of the tympanic cavity, the mastoid antrum, the mastoid cells, the internal ear, the internal auditory canal (as reported by Pierce and Toynbee), the aqueductus Fallopii, the carotid canal, by way of the foramina for nutrient vessels to the middle ear, communicating veins with the jugular fossa, the foramen for the auricular branch of the vagus nerve and the fissure of Glaser, which contains the tympanic branch of the internal maxillary artery, by the vascular interspaces between the unossified and the ununited osseous portion of the temporal bone, especially in infants, and by the small venulas traversing the mastoid region.

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[1] Prog. Med. Wochenschrift, 1891, No. 3.
[2] British Medical Journal, 1889, p. 615.

The site of necrosis and osseous perforation and subsequent infection may be through the tegmen tympani et antri and the sigmoid groove, but it is more frequently through the tegmen tympani et antri; thus, an abscess in the temporosphenoidal lobe is much more frequent than in the cerebellum.

Cerebellar abscesses are, according to Whitehead, more often due to disease of the petrous bone, generally involving the labyrinth, and less frequently due to sinus thrombosis.

The bibliographic references observed by the author show twice as many abscesses in the temporosphenoidal lobe as in the cerebellum. The above statistics may, however, be far from the truth, for Politzer quotes Körner's observation in one hundred cases in which 62 per cent. were in the cerebrum and 31 per cent. in the cerebellum, with a simultaneous involvement in 6 per cent.

Dr. Thomas J. Harris, in a review of 12,744 cases of suppurative ear disease in the Manhattan Eye and Ear Hospital, found that 60 cases suffered from meningitis, 23 from sinus thrombosis and 7 from abscess, in six of which cases the abscess was located in the temporosphenoidal lobe and one in the cerebellum.

Gowers says that suppuration appears four times as often in the cerebrum as in the cerebellum and rarely in the pons or medulla oblongata.

It was also interesting to note in the bibliography at my disposal that 30 cases of otorrhea cerebralis are reported; Hoffman reports one case in 1827, Albers in 1837, De Boun in 1838, Muckel in 1839, etc.

In regard to otorrhea cerebralis, Macewen says that a condition of this character might actually become established, provided one understands by that term leakage by pathological processes from an abscess in the brain, through a pre-existing osseous erosion, by which the infectious matter originating the abscess entered the cerebral tissue.

Gruber relates a case in which a cerebral abscess emptied into the middle ear, which, in his opinion, was previously healthy.

Among the intracranial lesions which may originate from the ear and its adnexa are tumors, cholesteatomata, acute or chronic cerebral or cerebellar abscesses, leptomeningitis, pachymeningitis, basilar meningitis, epidural abscess, sigmoid sinus thrombosis, jugular thrombosis, sinus phlebitis, cavernous sinus thrombosis, brain tumors, local cerebral softening, caseous degeneration, gangrene, thrombosis of the cerebral tissues, ulceration of the brain and primary bulb thrombosis. The last condition, however, is one open to question.

Among those who especially advocate the theory of primary bulb thrombosis are Randall and McKernon, who believe the disease to have extended to the bulb from the floor of the tympanic cavity, especially in children suffering from acute otitis media suppurativa, and is more often observed where the jugular bulb is in close proximity to the floor of the middle ear. The opponents to this theory believe that a macroscopical absence of disease in the mastoid process is no evidence that septic micro-organisms have not reached the sinus wall by metastasis, and that bulbar thrombosis is purely a secondary inflammation. Personally, I can see no anatomical or pathological reason to doubt the possibility of primary jugular thrombosis.

The character of infection extending from the middle ear varies according to the cause producing the ear lesion, and may be monobacterial or polybacterial. Intracranial lesions not alone originate from suppurative lesions of the ear. but may be due to infection from non-suppurative inflammation.[3]

The infectious organisms observed in brain disease of otitic origin are the *Streptococcus pyogenes aureus, Staphylococcus pyogenes aureus,* tubercle bacillus. Eberth's typhoid bacillus, Klebs-Löffler bacillus, the diplococcus intracellularis of meningitis, and the pneumococcus.

Allport[4] quotes von Bergmann as expressing the opinion that no brain abscess occurs without the presence of *Streptococcus pyogenes aureus.* Allport, however, does not accept this view. In one case observed by the writer,[5] abscess of the temporosphenoidal lobe contained only pure *Staphylococcus pyogenes aureus* in the pus.

In brain abscess we usually have one or both of the above organisms, sometimes combined with saprophytes, and the same may be said of leptomeningitis, pachymeningitis and sinus thrombosis.

As a rule, septic infection of the brain extends directly or indirectly from a suppuration in the middle ear; however, it must be noted that a primary inflammation of the mastoid cells may occur from metastasis or traumatism, as observed by Snydacker and others, and extends directly to the cranial cavity without involvement of the middle ear.

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[3] Donivan, Trans. Maine Med. Assn., 1884.
[4] Chicago Medical Record, 1906.
[5] Annals of Otology, Rhinology and Laryngology, 1906.

Macewen, in his valuable work on pyogenic diseases of the brain, asks the question, Can an abscess in the brain form from an infective otitis media purulenta and remain *in situ* while the middle ear disease becomes cured? and says such a condition is highly improbable, and reports a case by Gruber, witnessed by himself, which showed at least that healing had taken place in the external part of the ear while the abscess existed in the brain.

Matthewson[6] reports a case of abscess of the cerebellum, following otitis media, months after an apparent cure. In a case observed by myself the abscess in the temporosphenoidal lobe occurred nine years after all discharge from the ear had apparently cased. Macewen's question can thus be answered, I believe, in the affirmative.

Cerebral complications may terminate in resolution or in death. The most fatal of all cerebral complications from otitic infection is purulent leptomeningitis, and especially is this true where there is a general infection.

Infectious pachymeningitis and extradural, cerebral or cerebellar abscess usually recover if detected early and operative measures are instituted. The results in infective sinus thrombosis are often more favorable, especially if there exists no secondary infiltration in the lung, liver, heart or kidneys. Many cases of pyemia from septic sigmoid sinus thrombosis are reported as having recovered after operation. My results have been unsatisfactory. In five cases operated upon during the past two years four died and one recovered. One died of metastatic abscess of the lung, complicated with endocarditis, one from a general pulmonary metastasis and the other two from purulent leptomeningitis. These cases all followed acute suppuration in the middle ear.

The termination of a brain abscess, after the exciting cause has been removed, may be in a number of ways, such as rupture, flooding the meninges, absorption or encapsulation. Abscess cavities in the brain may rupture through the roof of the tympanum or mastoid antrum, as shown by Heath and also by Hawkins as early as 1835.[7] The abscess may drain through a fistula in the mastoid process external or internal to the process into the neck muscles. The early diagnosis and evacuation of pus by surgical measures is the ideal termination of an intracranial abscess.

Macewen has accentuated a point which may be opportunely referred to, and that is the danger which may follow the removal of polypi of the middle ear on account of the frequent connection of such growths with a suppuration in the cranium or meninges.

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[6] American Journal of Otology, 1882.

[7] London Mag. and Gazette.

Contour[8] early reports a case of abscess of the brain following polypus of the ear. Removal of polypi and granulation tissue may also permit new infection to pass through bony erosion, to the meninges. The literature on the subject contains a number of references to this point, as well as the danger of forcing fluids into the middle ear in cases of chronic suppuration and of having the fluids pass into the meninges or abscess cavity, thus producing an active inflammation and probably death.

It is beyond my ken to give hard and fast rules for differential diagnosis of intracranial lesions; the subject is too extensive for me to even begin, and in consequence any further consideration of the subject will be limited to meningitis and sinus thrombosis.

MENINGITIS.

Meningitis may imply pachymeningitis or leptomeningitis; however, the latter lesion is implied in a discussion of meningitis. Meningitis may be purulent or serous; on account of the varied degree of the latter lesion encountered originating in the ear, it is really of more prognostic interest than the cerebral or cerebellar abscess. The differentiation everybody admits to be frequently difficult.

Meningitis due to infection from the pneumococcus runs a much more rapid course—five to six days—than that due to infection from the diplococcus of Weichselbaum, or the tubercle bacillus, which may run a long course, varying from a week to two or three months. In favorable cases, however, the improvement usually begins after the first week. Cerebrospinal meningitis is presumed to be due to the diplococcus intracellularis alone. Meningitis may be due to single or double infection, which may be caused by the diplococcus of meningitis. the *Staphylococcus* or *Streptococcus pyogenes aureus,* the pneumococcus, typhoid bacillus, bacillus tuberculosis, gonococcus as observed by Trow as a cause of mastoiditis,[9] the Klebs-Löffler bacillus, the streptococcus of erysipelas, the grippe bacillus and unknown bacteria. The tubercle bacillus or the *Diplococcus intracellularis meningitidis* of Weichselbaum may multiply and remain with a suppurating ear, causing a condition of slumbering mastoiditis, which for years produces a slight irritation of the meninges, only awaiting an attack of influenza, pneumonia, typhoid fever or some disease sufficient to lower the resisting power of the meninges to bring about this severe complication.

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[8] Bull. Soc. Anat. de Paris, 1842.
[9] Canadian Pract. and Review, 1903.

Meningitis occurs more frequently in men than in women, the frequency of the disease having no relationship to epidemics of meningitis, but occurring at any time or season of the year.

The predisposing factors of the disease are a general lowering of the vitality of the individual and extension of suppuration, especially in the mastoid and labyrinth. Meningitis of otitic origin differs from the ordinary meningitis or cerebrospinal meningitis in that the disease is seemingly free from the tendency to spread to other individuals. The contagiousness or infectiousness of the disease of otitic origin has never been observed by the author, whereas in the idiopathic form of the disease its contagiousness has always been marked and every precaution taken to prevent its spread. The meningococcus may extend to the ear and meninges direct from the mucous membrane of the nose and throat of the individual, and in exceptional cases from the operator or nurses in attendance at the operation. It is a good rule while operating on the ear in severe suppurative disease to protect the faces of all present and actively engaged in the operation with moist sterile or bichlorid gauze.

The following analysis of a case will illustrate a condition found in slumbering mastoiditis and irritation of the meninges, at times sufficient to impair the memory and seriously retard the education of the individual because of the accentuation of the irritation upon close application to study.

Case 1.—Chronic discharge from the left ear for eight years; no odor to slight discharge which was detected glazing the inner tympanic wall.

Blood examination by Dr. W. T. Dodds:

Red cells	4,536,000
White cells	10,000
Polymorphonuclear variety	8,800
Mononuclear variety	600
Lymphocytes	400
Transitionals	200
Hemoglobin	393
Color index	102

Microscopical examination of pus from middle ear: Large cuboidal epithelial cells; many pus and mucous corpuscles; staphylococcus in abundance and a *Diplococcus intracellularis meningitidis,* the latter being in the pus cells.

Urine.—Two per cent. urea, excess of chlorids, uric acid and urates.

The above examination with subjective symptoms showed necrosis in flat bone and general absorption, with a disposition to cerebral complication. Meningitis serosa is but a short step from meningeal irritation due to middle ear suppuration. The serous meningitis of Quincke in complication with acute otitis media or accompanying or following a mastoidectomy, is an especially interesting condition, first, because of the alarming symptoms, and second, because of the favorable outcome, many times, of the disease. Serous meningitis of Quincke is suddenly, during the otherwise seemingly satisfactory course of the disease, ushered in with a high temperature, rapid pulse, delirium and symptoms of pressure in the posterior fossa. There may be present optic neuritis and strabismus.

In one case observed by the author, one week after simple mastoidectomy following acute otitis media purulenta, there was delirium for two weeks, with a temperature varying from 100° to 105.° In this case no operative measures were resorted to and the patient made a perfect recovery. Cerebral symptoms of pressure were present though not profound. There were present high temperature, delirium and a rapid pulse, though no optic neuritis. A condition of this character was evidently metastatic and due to the organism producing the mastoiditis, the *Streptococcus pyogenes aureus* or its toxins. The blood examination showed a high leucocytosis, with abundance of polymorphonuclear leucocytes. In this case a purulent discharge persisted from the mastoid wound for two months, completely disappearing with perfect healing of the wound.

According to Quincke, any operation made into the arachnoid space will be followed by a cure.

Ballance, in a discussion of a paper by Dench on treatment of intracranial complications of middle ear suppuration, read before the Section on Laryngology and Otology of the American Medical Association, June, 1906, said there is a rise of temperature, slowing of the pulse, vomiting, dizziness, stability of the pupils and some redness of the optic disc, and that the condition can be relieved at once by lumbar puncture. Dench (ibid) said: "I am inclined to believe that the best plan of treatment is to relieve the pressure by lumbar puncture and, failing in this, to relieve the increased pressure by craniectomy, followed by an incision of the dura and of the cerebral substance." He also said he believed it better, provided the ventricular distension was not sufficient for free drainage after the brain substance had been penetrated for the distance of an inch, to simply incise the brain substance.

In addition to the symptoms enumerated by Ballance, the general symptoms of meningitis are headache, delirium, muscular twitching of arms and legs, grinding of the teeth, involuntary discharge of urine and feces, contraction of the muscles of the neck, contraction of the pupils, injected conjunctiva and hyperesthesia of the muscles of the neck and calves of the legs. In very young children epileptiform convulsions and opisthotones may be the first symptoms of the disease, with now and then a peculiar "hydrocephalic" cry. The temperature may vary from 100° to 106° and the pulse from 50 to 120 per minute, with a tendency to drop to the lower figure.

Kernig's sign is usually present, i. e., an inability of the patient while lying on his back to flex the thigh without at the same time flexing the leg, and also the inability to completely extend the leg.

Trousseau's sign may also be present, as shown by the contraction of the muscles of the upper extremities, following pressure on the tendon of the biceps.

In enumerating the eye symptoms observed in meningitis I can do no better than report the compilation on the subject by Robert B. Preble:[10] "Heine reports a study of the eye disturbance in epidemic meningitis based upon a study of 100 cases. Disturbances may appear in the motor apparatus, the sensory tracts or the nutrient portion of the eye. One should not include here the cases of conjunctivitis, keratitis and the like, which sometime develop in the course of this, as in other severe infections, when the patients lie unconscious with half-open eyes, for a long time.

"Hemorrhages into the retina are seen very much less often than simple intraocular optic neuritis. This is more often due to a descending neuritis from the basal meningitis. Sometimes there is a neuritis of the optic nerves behind the bulbs and without ophthalmoscopic findings, unless optic atrophy results. The frequency of secondary optic atrophy is not yet determined for want of sufficient statistics. Metastatic ophthalmia is seen and is usually severe.

"Involvement of the motor oculi nerves also occurs. They are almost always basilar in origin, more often unilateral than bilateral, and oftenest the abducens paralysis. Not rarely one sees a more or less complete paralysis of all the eye muscles. Ptosis is strikingly often absent. The milder cases often recover completely.

"Of the 100 cases examined by Heine 20 showed eye symptoms which totaled 30 in number. There were 9 cases of unilateral or bilateral optic neuritis or retinitis, 13 with motor oculi paralysis and 5 with ophthalmia."

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[10] Progressive Medicine, vol. iii, No. 1.

Blood examination in meningitis shows a high leucocytosis, varying from 40,000 to 47,000 (Emerson), and according to Preble there is no material difference in the counts shown by the cases ending fatally and those which recovered.

Lumbar puncture in meningitis serosa or purulenta is a proper procedure for two reasons: First, as relief of the pressure in the subarachnoid space and ventricles, and second, as a diagnostic measure. The fluid, as described by Schottmüller, may be perfectly clear in meningitis, ranging from that of a cloudy or purulent fluid. The cocci vary in number, the number having no relationship to the color of the cerebrospinal fluid. In brain abscess the cerebrospinal fluid is clear and free from cocci.

According to Lenhartz, the amount of fluid to be withdrawn is from 25 to 40 c.c. at each sitting and should be performed daily.

SINUS THROMBOSIS.

Sinus thrombosis may involve the lateral, sigmoid or jugular bulb. All three divisions may be involved at the same time or the inflammation may confine itself to one structure, beginning at the torcula and extending the entire length of the jugular vein of the affected side. Other sinuses may become involved secondarily from metastasis.

In diseases of the jugular vein and sinus thrombosis there may be present metastatic infiltration in the lung, liver, kidney and heart.

The disease is usually ushered in by rigors, which increase in number and severity and are followed by profuse sweating. The temperature is very high, differing thus from meningitis, ranging from 100 to 106° F. The pulse is exceedingly rapid, and as the infection increases becomes weak and thready. There is rapid emaciation and toward the fatal termination of the disease involuntary evidence of urine and feces. Abscess of the lung or liver accompanying the disease may rupture, causing the sudden death of the individual.

Early in the disease there is pain in the region of the thrombus upon pressure or percussion. Though this symptom may be entirely absent, we not infrequently have tenderness upon pressure, swelling of the tissues over the mastoid and muscles of the neck. The important symptoms differentiating sinus thrombosis are severe chills, followed by profuse sweating, absence of the characteristic pain in the ear and head, as observed in meningitis and brain abscess, and high temperature. lower in the morning and higher in the afternoon. The intellect is clear during the early stage of the disease and before profound pyemia is established. The disease usually terminates in from two to three weeks.

Blood examination shows a high percentage of polymorphonuclear leucocytes.

The treatment of the disease varies somewhat, according to the extent of the lesion. My observation is that sinus thrombosis is more often fatal when the necrosis has extended into the petrous portion of the temporal bone. With a deep necrosis in this region other complications are more likely to occur.

Leucocytic count shows a high degree of leucocytosis, varying from 10,000 to 40,000. This condition might also be expected in acute abscess of the mastoid process; however, a high leucocytosis is indicative of greater extension of suppuration than in the mastoid alone. The blood may show streptococcic or other infectious organisms. A leucocytosis of 10,000 or more, following a few hours or days after operation and sustained for any length of time, is indicative, according to Emerson, of deeper complications.

At present the question of most importance is, shall the jugular vein be ligated in all cases of sinus thrombosis?

Whiting[11] "considers that the inability to re-establish the circulation from the bulb was a positive indication of resection of the jugular, whether the clot contained at the bulb has undergone purulent disintegration or not; also that the jugular should be resected in every instance when the clot in the sinus has undergone purulent disintegration."

John D. Richards[12] says that primary jugular ligation or resection is rarely indicated except in the presence of metastasis.

Kennon[13] says, should a thrombus be situated either primarily or secondarily in the bulb, we should at once expose and resect the jugular vein.

Dench[14] says: "Sinus thrombosis recognized early, will be almost certainly cured." Dench further says (ibid). "In a case where the general condition of the patient is good, jugular exsection should be performed at the first operation if full hemorrhage does not take place from the bulbar end of the sinus."

Before resorting to the complete exsection of the jugular vein it is well to consider, first, whether or not we have any metastatic complications, as pneumonia, abscess in the lung or septic endocarditis. With such complications, severe in character, there can be no good derived from a primary resection of the jugular vein. Even in a consideration of primary jugular exsection there is the constant possibility of purulent phlebitis and infective embolic substance passing through the collateral circulation in the neck. In the present light I should prefer to explore the sigmoid sinus and with the thorough establishment of circulation and absence of purulent phlebitis, plug the wound, finding the bulbar region involved with a retarded flow of blood from above or below and the patient strong, would recommend jugular resection, resecting the vein as completely as possible.

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[11] Archives of Otology, June, 1906, p. 253.
[12] Archives of Otology, June, 1906, p. 378.
[13] Archives of Otology. June, 1906.
[14] Journal A. M. A., October, 1906, p. 1290.

DISCUSSION.

Dr. Sprague (by invitation):—I thank Dr. Ballenger for asking me to take part, but I came as a student, and the papers have been so complete that I see nothing to add, except perhaps two points. One is with regard to diagnosis from the mental symptoms. I have seen in one or more cases diagnosis from the mental disease where the mental symptoms were not those of any form of insanity. In fact, I have never seen in any of these cases under discussion mental symptoms which resembled or were even so closely similar as to deceive the neurologist into the belief that it was a mental disease rather than a mental symptom. The other point is that in many of these fatal cases we have seen it may be charged to faulty technic—not in surgery, but in the application of the knowledge we have. If the symptoms related here to-day were attended to in all these cases the mortality would be greatly reduced. We do not examine the eye. We do not instruct the general practitioner or warn the patient or his relatives sufficiently as to the subsequent danger of a purulent ear. We do not pay attention to early mental confusion. We do not look into the blood count or sufficiently care for the temperature, pulse, etc. The most important point to be remembered, coming out of such a discussion as this, is to be careful about the diagnosis.

Dr. Andrews:—The experience which I have had in two or three cases leads me to believe that all external indications of an ear disease may disappear and still there be present a brain lesion. I remember one case where there had been no history of discharge or other trouble of the ear for a number of years, and still we had a mastoiditis and a brain abscess. I think it exceedingly probable in this case as well as the other cases to which I refer, that there was a discharge from the brain abscess through the tegmen antri, the middle ear, and the Eustachian tube, and that it was not apparent to either the patient or the physician under whose care the patient had been. I think now of three cases where we had mastoid disease and brain abscess, without any discharge from the ear or any indication of middle ear trouble for a considerable number of years previously.

Dr. Stein, Chicago:—This paper recalls to my mind a case that I reported before this society when we met in Chicago some six or seven years ago. The case is on record, but I do not recall all of the details. It was a child, 2 years of age, that had various symptoms that ultimately resulted in the diagnosis of tubercular meningitis. The child was first under the care of its family physician, and later five or six other doctors, including several specialists—I mean nerve specialists and children specialists—and the final diagnosis was that the child had tubercular meningitis. The prognosis naturally was that the child would die, and in looking at it, one certainly would have entertained the opinion that the child was about to die. At this stage I was called in. I was called in for the reason that the family doctor remembered that I had seen this child when it was six or eight months old with an acute middle ear trouble, but which soon disappeared. I remember incising the drum membrane at the time. I thought possibly there might be something the matter with the ear, although there was no evidence externally in any way to indicate that there was ear trouble. While I was examining the child, with everybody standing around and thinking every moment the child was going to die—and I certainly thought so myself—and after examining one ear, and in turning it over to look at the other ear, we noticed the pillow was wet—saturated with pus, which was coming from the auditory canal. I looked at the other ear, and it looked to me as if there was something back of it. I incised it and pus came out of that middle ear. This child ultimately recovered. I reported this case as a double-sided extradural abscess—for the want of an autopsy, because the child recovered—but we discussed it at that time, and no one contradicted my diagnosis, and it has been put on record as such. Now it is intensely interesting to recall this case at this time, particularly because of Dr. Andrews' remarks. I think that child had trouble slumbering in that ear for the year and a half following the time I had seen it, and it suddenly developed some intestinal disorder, which the family physician said surely existed at the outset, and that probably was the factor to bring forth the slumbering trouble which lay between the floor of the middle fossa and tegmen of the middle ear, and the meningeal symptoms—the local meningitis—was manifested in what was finally diagnosed as tubercular meningitis, but fortunately, as the result of spontaneous rupture, the child recovered.

Dr. Ballenger:—I want to briefly mention one or two cases of slumbering ear disease, of which Dr. Kyle has so well spoken. I remember one case which eventuated in a cavernous sinus thrombosis. I have been so unfortunate as to have had two cases of cavernous sinus thrombosis in my own practice, both of which proved fatal. One patient, to which I refer now, at 4 years of age had scarlet fever, followed by otitis media purulenta, which, according to the family, ceased after a few weeks, and for seven years this little girl had no apparent disease of the middle ear. I do not believe it. Seven years later she had an acute exacerbation, followed very quickly by involvement of the lateral sinus and the cavernous sinus, probably extending to the cavernous sinuses through the superior or inferior petrosal sinuses. At first one eye was involved, and later the other eye by extension through the circular sinus. Here was a case apparently well for seven years which later developed a sinus thrombosis. I had another case in a little boy —I forget how many years his disease had been cured—using the word "cured" in quotations. Anyway, when I saw him he was in a comatose condition and his hearing was destroyed, as the labyrinth was involved. The drumhead was not perforated; slight tenderness over the mastoid antrum, at least I thought I could detect slight pain in that region. I incised the drumhead, and there was a discharge of pus. I placed the boy in a hospital and operated on him that night, and he died of meningitis a few hours later. Now, here are two cases that were apparently cured for years. "Cured" is a term we should use with caution in our vocabulary in reference to infectious diseases of the ear. It takes the observation of an expert to determine whether a case is cured or not. One of the best evidences of a cure, perhaps, is continued closure of the perforation in the eardrum. That, to my mind, is one of the best evidences of a cure, though even this is not positive, as I have shown by my cases. Ordinarily, however, this would be a fair evidence of cure. If the drumhead is still perforated, and the secretion is so slight as not to discharge into the external meatus, we cannot say positively that the case is cured. Dr. Bryant, who has a new method of curing ear diseases—or an old method—can say something, perhaps, on this subject.

Dr. W. Sohier Bryant:—I do not know what I can say in review of Dr. Kyle's paper, except to add my own experience in the same line. We have all seen these cases apparently cured of middle ear diseases with serious complications following some time after without any evidence of recurring disturbance in the middle ear. I think the general practitioner has never been acquainted with the possibility of such occurrences, and that is why the otologists as a rule are not called in time in such cases. An important point, as suggested by one of the doctors, is that an otologist should be called in to pass on such cases some time after they have apparently healed. Where the drumhead is healed there may be some irritation inside which the general practitioner, though familiar with otoscopic examination, would not be qualified to pass judgment on. The question of treatment—the prophylaxis of mastoiditis—was spoken of several times in the papers this morning. I think an early operation in cases of mastoiditis is what we are tending towards. As the improved technic and better results get more widely known, we can more forcibly assure quick and successful recovery of the patient. We have already found that convalescence will be shorter, and the constitutional disturbance much less and the functions better preserved by an early operation than by a longer and conservative treatment—which often results in what you might call a late operation.

Dr. Beck:—Anticipating that in the papers read on suppurative ears there would be some discussion on the lumbar puncture, I went to the trouble of looking up cases that were so treated in the Cook County Hospital since Quincke devised this operation. There have been forty-nine cases so treated for different purposes, thirty-two of them in cases of suppurative trouble in the ear, complicated with meningitis, or purulent meningitis cases. It is interesting to read the histological reports in the cases, and only in about 2 per cent. did they find micro-organisms, but in 82 per cent. of these forty-nine cases they found cloudy fluid, which was due to the marked increase of leucocytes, so I would call attention to the fact that if this modern procedure is carried out for diagnostic purposes, and you draw your fluid and find it turbid, you must not conclude that you have infected material, though the increased pressure is usually associated with a marked increase of leucocytes in this fluid. I hope to be able to show one plate that will demonstrate a clear middle ear mastoid, and also show the rest of the intracranial structures. Of course, I have no plate to show a tumor or abscess, but certainly it could be demonstrated. I think the radiographs should aid us a great deal in establishing the fact whether we have a condition healed, even in spite of the fact that your drum head is healed over.

Dr. Dean:—I should like to ask the members of this section in regard to a case which I had. The patient was a boy, 8 years of age, who had a slight otitis media, the result of tonsillectomy. The ear trouble developed three weeks after operation. I saw him a week after this; when he had a septic temperature and had a cord-like swelling on the right side of his neck just anterior to the sterno-cleido muscle. There was a slight discharge from the ear. On the left side, exophthalmos and panophthalmitis; on the right side, optic neuritis. At the time, I made a diagnosis of septic thrombosis of the lateral sinus and advised an operation, which was refused. The patient recovered, apparently none the worse, except for the loss of the right eye and a diminished vision in the left. Several weeks ago I saw an abscess of the brain on the left side, present three months after the patient had measles. The members of the family and the family physician said there had been no discharge from the ear for a period of two months, and at the time I saw the patient, there was apparently no fluid in the tympanic cavity.

Dr. Kyle (closing):—I do not know that I have anything to add to the discussion. I think that we should keep our cases that have apparently healed under observation for quite a while, even after the discharge has ceased, and I think Dr. Ballenger has accentuated the point that these cerebral troubles may arise from an old rupture in the membrane. That the organisms grow within the middle ear seems certain, and though not of a virulent character, yet strong enough to keep up a mild inflammation, and under climatic influences we may produce a suppuration with involvement of the mastoid process and brain. I do not know whether I can answer the doctor's question as to that case. I should think that he had a serous meningitis with infiltration of the lymphatic glands, probably both due to the tonsillitis.

EMPYEMA OF THE SPHENOIDAL SINUS.

ALBERT H. ANDREWS, M.D.

CHICAGO.

In studying the general anatomic arrangements of sphenoidal sinuses in a considerable number of cadavers one observes several interesting points:

1. The cavities are situated in the angle formed by the junction of the nearly horizontal roof of the nasal cavity with the nearly perpendicular posterior nasopharyngeal wall.
2. The irregular shape of individual specimens and the lack of symmetry in the two sides.
3. The irregular arrangement of the sphenoidal septum and that its irregularities are independent of any irregularities of the nasal septum. Sometimes the nasal septum being deflected well to one side and the sphenoidal septum well to the other. In other cases the septum is placed diagonally between the two sinuses.
4. The frequency with which one of the large posterior ethmoid cells invades the space usually occupied by the sphenoidal sinus.
5. The thinness of the bone forming the upper part of the anterior wall, sometimes the bone being entirely absent.
6. The close proximity of this cavity to the cranial cavity, to the orbit and to important blood vessels.
7. The varying position of the ostium in relation to all the surrounding landmarks.
8. The high position of the ostium interfering with drainage should the cavity become diseased.

In looking over the literature of sphenoidal empyema one notes the small amount of space devoted to the subject by the older authors and the considerable number of articles appearing recently especially bearing upon the relation of sphenoidal empyema to diseases of the eye.

In presenting these notes, preliminary to a more extensive paper in course of preparation, it is the writer's purpose to call attention to a few points in the diagnosis of sphenoidal sinus disease and to touch but briefly upon the subject of treatment.

The cases naturally divide themselves into two classes, acute and chronic, neither of which present subjective symptoms of much value in differentiating empyema of this sinus from some of the other accessory cavities of the nose. In both the acute and chronic cases the accumulation may be either pus, mucus or a combination of the two.

The one objective symptom which should call for careful investigation is the presence of pus or mucus in the space above the posterior end of the middle turbinate. A discharge from this locality may come from the surface of the upper meatus, the posterior ethmoid cells, or from the sphenoidal sinus. A plan which in several cases has given evidence of the source of the discharge is to push as large a pledget of cotton as possible into this space, and then after the patient has held his head in a variety of positions, especially forward, to remove the cotton and note if any part of the pledget seems to have been against a discharging aperture. Ordinarily the cotton is saturated with a cocain solution to prepare the tissues for further examination. The pledget is applied on a bent aluminum applicator and the applicator is left in position until the cotton should be removed. Frequently the cotton will show unmistakable evidence of having been in contact with a discharging sphenoidal foramen. The use of one of the various forms of suction apparatus will also give valuable additional evidence not only in these cases, but of empyema of the other accessory cavities.

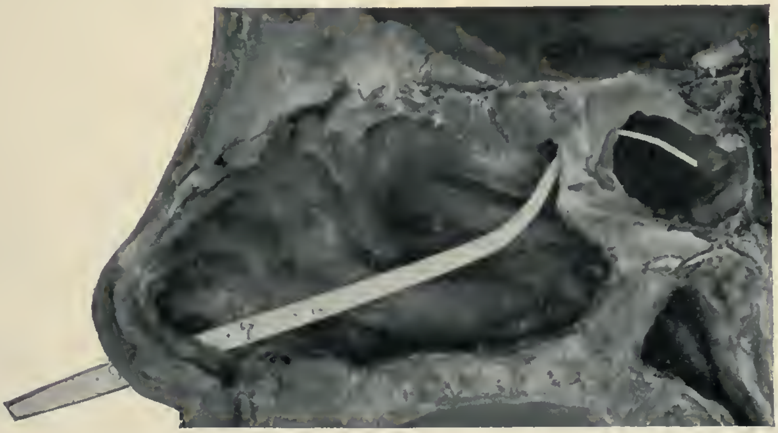

Fig. 1.—Sagittal section of nose with septum removed, showing probe passsed through ostium.

Of the direct methods the probe and the aspirator give most reliable evidence. After removal of the middle turbinate it is usually easy to probe the sinus, but one frequently suspects that the sinus may be diseased when he would hardly feel justified in removing the turbinate in order to determine this. After studying the relation of these parts in more than fifty cadavers and exploring the sinuses in as many patients the writer has no hesitancy in saying that this can usually be accomplished without removal of the turbinate, even though it may lie very close to the septum. The probe for this purpose must necessarily be slender and should have a double bend, first upward about five centimeters from the end and then downward again until the end is in line with the shaft of the probe. In addition to this bend it is frequently necessary to give the probe a slight lateral bend away from the septum.

The elevation of the bend above the line of the shaft of the probe is about two centimeters. While the shape described will not always enable the operator to enter the sinus, it is a good thing to begin with, and the probe can be reshaped as the necessities of the case arise. Flat graduated probes answer the purpose nicely.

These probes are marked with the centimeters and half centimeters from six up to eleven. Because of bending the probe near the end the marking may not be accurate, but since the object of the markings is to facilitate the comparing of distances rather than the making of accurate measurements, this lack of accuracy has no importance. The canula used for aspirating and also for washing out the sinus is flat and shaped similarly to the probe described.

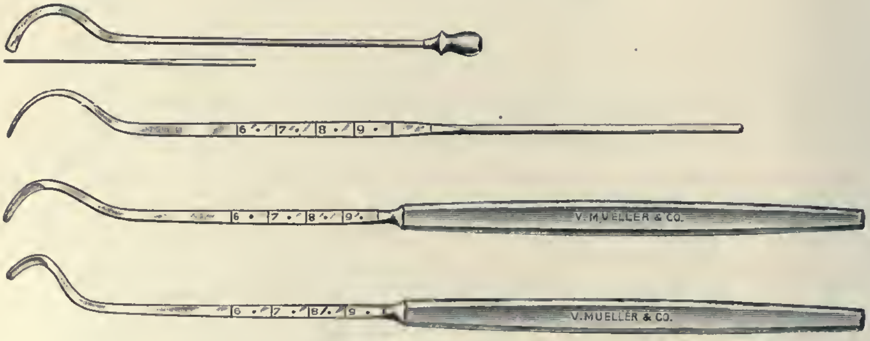

Fig. 2.—Canula for aspirating and treating cavity. Probe for locating ostium. Knives for incising an anterior wall of sinus.

The position of the ostium close under the roof of the nasal cavity, together with the backward and downward direction necessary to enter the sinus, makes this form of probe best adapted to exploring this cavity.

The writer's rule for ascertaining when the probe has entered the sinus is to compare the distance it has entered the nasal cavity with the distance directly backward to the posterior pharyngeal wall.

In the application of this rule first insert the probe straight backward until the end comes in contact with the posterior wall and then mark the probe where it enters the nostril. With the graduated probes this is easy to note, and when the plain probe is used it may be marked by drawing some smooth hard instrument across it until it is marked at the point where it enters the nostril. The probe should now be passed backward and upward between the middle turbinate and the septum and search made for the ostium. If the probe enters the nostril as deep in this direction as it passed directly backward the evidence is sufficient that it has entered the sphenoidal sinus. In the examination of the above mentioned cadavers this rule was found approximately correct in all cases.

In cases where we have reason to suspect the sphenoid and cannot locate the ostium we are justified in making an opening into the sinus for exploratory purposes. This should be high up in its anterior wall where the bone is thin and sometimes absent. The opening may be made by means of a stiff probe or a knife with a bend such as has been described. With such an instrument it is next to impossible to do any harm.

After the ostium has been located or a new opening made and the question of a diseased sinus settled in the affirmative, the question of treatment becomes important. As is the case in other accessory cavities, there are varying degrees of pathology, and the treatment which will be efficient in one will not be sufficient in another, and the treatment which will be necessary in one will not be justified in another. In the severer case removal of the middle turbinate and extensive removal of the anterior wall of the sinus are required. After removal of the middle turbinate this can readily be accomplished with some of the cutting forceps.

In the milder cases cleansing the cavity a few times either with some antiseptic cleansing agent or with the air douche is all that is necessary. I have shaped a canula which I have found valuable in aspirating and in treating the sinus. It is flattened in order to pass easily between the middle turbinate and the septum. I have also made some knives with which I have been able to enlarge the opening by incising the anterior wall. In the cadaver I have usually been able to make a slit one to one and a half cm. long. In the patients I have usually been able to enlarge the opening sufficiently for purposes of cleansing and medication.

In conclusion let me state that in this work two things have especially impressed me. 1. The number of cases of sphenoidal diseases I have found since I have made these examinations a part of my routine work, almost all the cases which were formerly called "postnasal catarrh" now being shown to be empyema of some accessory cavity, most frequently the sphenoidal. 2. The ease with which the cavity can be explored and its condition determined when suitable instruments are used and the efforts are properly directed.

RADIOGRAPHY AND TRANSILLUMINATION IN DIAGNOSIS OF SINUS DISEASE.

JOSEPH C. BECK, M.D.

CHICAGO.

The value of radiography for diagnostic purposes is so well established at the present time, especially in connection with general surgery, that I feel it is unnecessary to take up its origin and progress. However, so far as its application in our specialty is concerned, I must admit that it is very much neglected. The cause, I believe, is due to the fact that the results thus far obtained have been very unsatisfactory. The blame for this can be laid to the radiologists, most of whom are not familiar with the anatomical structures of the sinuses (Grashey's Atlas[1]), nor their pathological lesions. Consequently the results obtained are unsatisfactory in most instances, and there is great lack of enthusiasm on the part of the radiologist as well as the rhinologist. I am convinced that if a radiologist were to make himself acquainted with the above conditions, namely, anatomy and pathology of the sinuses, he would obtain just as good results in this line as he does from skiagraphs of stones in the kidney, diseased lungs, etc., and would aid us a great deal in the diagnosis and treatment. Under the present conditions I believe the best remedy is that the rhinologist assist the radiologist in obtaining the best results. The former is to outline the disease, and the latter does the mechanical part of taking the radiograph and developing it; finally, both study the plate and come to conclusions. That has been my method of procedure.

Albers Schoenberg[2] is one of the few radiologists abroad who has devoted considerable time to developing the radiology of the head, but in this country the credit belongs to Caldwell, who, with the association and cooperation of Coakley, has given us a valuable method of obtaining good plates of the sinuses. The only regret I have is that for such a long time was the method known to them and not published, although presented to a society of radiologists for a year past; that much valuable information could have accumulated by this time on the radiography of the sinuses. I have been familiar with a number of rhinologists who also have spent a great deal of time and money on radiographs of the sinuses, only to be disappointed by their results. This, I say, was all due to imperfect technic in not knowing exactly at what angle the rays should traverse the head in order to get a distinct image of the sinuses as to size and pathological condition.

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[1] Grashey: Atlas Typischer Rontgenbilder.

[2] Albers-Shoenberg. Atlas.

Scheier,[3] in 1897 and 1898, was the first man who demonstrated pus in an antrum by *x*-ray plate, in that he washed out the pus, filled the cavity with water, and showed an entirely different density in the second plate.

Winkler and Brautleicht[4] made a number of observations on *x*-ray plates taken of skulls devoid of soft parts, but most of their work was done in the transverse view. It was done with especial reference to Winkler's osteoplastic operation on the frontal and ethmoidal sinuses. Mosher[5] has made a number of observations on *x*-ray plates, and deduced from them certain measurements of the sinuses. Coakley,[6] in 1905, was about the first in this country to describe and give his results with the use of the *x*-ray in diagnosis of sinus disease, but so meager was his report as to the technic that not much knowledge could be obtained from his description. His radiologist, Dr. Caldwell,[7] who, I believe, does the most superior work in this or any other country, read a paper on skiagraphy of the sinuses, but his article did not appear until nearly a year later. Killian and Goodman[8] published their series of experiments on the skiagraphy of sinus diseases only last April, although their work precedes that of Coakley, the latter following their methods. Killian's results are very satisfactory, and his 38 cases that he reports were practically in the pathological condition and anatomical formation as the skiagraphs indicated. Their method in the technic is practically the same as I use and will describe presently.

The most recent work published by a rhinologist on the use of the *x*-ray for sinus diseases is that by Wassermann,[9] and, since his technic, results and conclusions are practically the same as mine, I, therefore, will not go into the details of his article, but present my personal experience and observation, which date back about a year. If I go into greater detail in the technic than necessary for the average radiologist, I hope to be pardoned, as I am speaking principally to rhinologists.

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[3] Scheler: Archiv. f. Laryngol., Bd. 6, Heft 1, 1897.

[4] Winkler: Fortschritte aud dem gebiete der Rontgenstahlen, Bd. 5, Heft 1, 1901, Bd. 6, 1902-3.

[5] Coakley: Annals of Otol., Rhin. and Laryn., 1905, vol. xlv.

[6] Mosher: Laryngoscope, 1906.

[7] Caldwell: American Quarterly of Roentgenology, January, 1907, vol, 1, No. 2.

[8] Killian and Goodmanns: Beitrage z. Klin. Chir., Bd. 54, Heft 1.

[9] Wasserman: International Centralblatt f. Ohrenheilkunde, Bd. 5, No. 10, July, 1907; and more extensively in Fortschritte auf dem Gebiete der Roentgenstahlen, 1907.

TECHNIC.

(*a*) *Equipment.*—1, Coil and cord; 2, table; 3 compression apparatus; 4, one medium tube; 5, high tube; 6, plates, 8x10; 7, developing room, with equipment; 8, transillumination box.

In case stereoscopic views are taken, one needs an apparatus for the examination of such plates, which is a large stereoscope.

(*b*) *Method of Procedure.*—In taking a picture of the sinuses in the anteroposterior direction, the patient is placed on the table, face down on to a plate, 8x10, in such a manner that the whole top of the head will be reproduced, also the lower margin to include the upper teeth. In that way all the anterior group of cells, frontal, ethmoid and antra, on both sides, will be represented. The second step is to adjust the compression apparatus in such a way that when the tube is put in the proper place over the compression apparatus the rays from the target, which is at a distance of eighteen inches, will pass above the occiput and not through or below it, as was formerly done by many. This is the essential point in the technic that I wish to bring out, for it is to avoid the dense part of the occipital bone and base of the skull, also in a measure will exclude the large sphenoidal sinuses that are liable to mask the other anterior groups of cells, as already mentioned by Coakley in one of his cases.

Caldwell describes a specific method in obtaining the proper angle by taking the base line, which represents the base of the skull, from the external auditory meatus to the glabella, and a line drawn at an angle of 25 degrees to this latter line gives the proper angle. The third step is to call attention to the patient, not to be startled when the current is turned on, but to remain perfectly quiet, although he can not move his head, anyway. It has been the practice of our radiologist to show the patient what the *x*-rays are, and this has always a quieting effect on neurotic individuals. The current is turned on, 110 volts, 80 amperes, and timed. The length of time we have varied according to the thickness of the skull and the kind of tube we use, whether high, medium or low. As a general rule, with a medium tube, we expose the patient from 60 to 80 seconds, and with a high tube from 30 to 40 seconds, in ordinary thickness of the skull, but in very massive skulls as long as two and a half minutes. The fourth step is to remove the compression apparatus, relieve the patient, take charge of the plate for developing. I might say that, for the protection of the plate from perspiration, it is a good idea to place between the plate and the patient an extra layer of waxed paper, else the plates, although wrapped in the double envelope, may be spoiled. Fifth, developing the plate. We use solution, about twenty minutes. The under-development of the plate is a very serious mistake in the technic. Permit to dry thoroughly from six to eight hours. Sixth, the examination of the plates, preferably in a darkened room, by means of a transilluminating box. With the aid of the radiologist, we study the outline and changes in the degree of cloudiness or density compared to the normal condition. This is the most interesting part to me, and to compare the results to other methods of examination, as transillumination and intranasal inspection.

In taking a skiagraph of a profile, that is, transverse view, the steps are as follows: First, patient on the side, depending on which side you wish to bring out most clearly. For instance, a right-sided frontal, ethmoidal and antrum or sphenoid disease, we put our patient on the right side, as the plate will show most clearly the structures nearest to it. Observe the same rule as to the head covering the entire plate. Second step, compression apparatus. Place so that the rays from the target pass through the base of the skull. This view will also represent a lateral view of the mastoid and middle ear. Third step, turn on the current and expose again, the time depending on the thickness of the skull and the degree of the tube. It requires a shorter exposure for transverse views, which are usually exposed from 40 to 60 seconds, with a medium tube. The fourth, fifth and sixth steps are the same as in the anteroposterior exposure.

If one wishes to obtain plates of the antra alone, one can take each side separately for stereoscopic views.

DEMONSTRATION OF NORMAL AND PATHOLOGICAL PLATES OF THE SINUSES.*

Plate 1.—Shows a skiagraph of a skull taken in the anteroposterior direction. It demonstrates the parts very clearly, owing to the absence of the soft parts. The contrast shows all dense substances white and spaces dark. The degree of white or black is significant in diagnosis of disease, or absence of the same. The anatomical landmarks are: (1) The orbita, with the superior and inferior margins; (2) the nasal spine and the lateral wall of the nose, as well as the floor, indicating the nasal fossa, divided by the septum; (3) malar bones; (4) superior maxilla, its alveolar processes and teeth; (5) the rami of the lower jaw. The details are: First, the frontal sinuses, with the various subdivisions or septa, on the left side, and a very interesting fact in this skull, discovered only after the plate was taken, i. e., the absence of the right frontal sinus, only showing the small diploic space. not communicating with the nose in the right side. Second, the ethmoidal cell, with the

lacrimal canal in front of it. Third, the antrum of Highmore, in which are seen many white streaks, representing the dense part of bone of the skull, as the pterygoid plates, etc., but a distinct outline of the wall of this sinus is very clearly shown. You can also observe some other anatomical points not seen when the soft parts are present. (a) Sphenoidal fissure in the orbita; (b) infraorbital foramen; (c) lines indicating the course of the blood channel; (d) sphenomaxillary fossa; (e) the two brass springs are shown most clearly; (f) the black line indicating the horizontal cut of the calvarium usually found in the preparation of a skull.

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* Owing to the unsatisfactory reproductdion of the plates for publication, the author decided not to use them, rather than to retouch them for such purpose.

Plate 2.—Same skull taken in the transverse meridian, with the right side next to the plate. It shows the same structures from the side, but not so clearly, however, inasmuch as they are superimposed one on the other. In addition, one can make out the sphenoidal and posterior ethmoidal sinuses and the depths of the antrum. You observe the absence of the frontal sinus, which is the right side. There is no frontal sinus on that side in this skull. Again, you observe the dense base of the skull, with the temporal bone. Here the mastoid process with the middle ear cavity is clearly demonstrated.

Plate 3.—A normal head, taken in an anteroposterior direction. This man has never had any nasal obstruction, not even a coryza. He has, in fact, never been ill, and is a perfectly developed man, and had as nearly normal nose, throat and ears as one can find. You observe the same anatomical conditions as in the skull, except not so clearly defined, on account of the soft parts. It, however, represents nearer the appearance of the structures in the study of disease. You observe the uniformity of the shadow of the cavity. The most important point in diagnosing obstruction or pathological changes of the sinuses from normal or thickening of the bone.

Plate 4.—Same head, taken in a transverse direction. Same conditions prevail as in Plate 2, of the skull, except the frontal sinus is clearly demonstrated. It shows its anteroposterior diameter, as well as its height, but you can see the superimposition of the two frontal sinuses.

Plate 5.—A case of double subacute sinuitis involving all the sinuses, following an influenza. All the usual clinical phenomena present, as pus from the various regions of the sinuses; tenderness on pressure, and the subjective symptoms, as morning periodical headaches and marked mental depression, are present. Transillumination is very unsatisfactory in this case, as both sides are involved. However, the light reflex can be made out in the pupillary area. The patient can not bear the pressure over the frontal sinuses

caused by the transillumination apparatus. The essential point in this plate is the cloudiness of all the anterior group of cells. The patient is under treatment only three weeks; local irrigation in the region of the ostea with normal salt solution, and is improving very rapidly. The showing of the plate after recovery is necessary to complete a complimentary picture of this plate, and will follow before publication of this paper.

Plate 6.—Double chronic frontal sinuitis of right ethmoidal, antral and sphenoidal sinuses. This plate shows a marked cloudiness of these sinuses, as well as all the other clinical phenomena, as severe headache, especially over the left side, are present. There is pus from the regions of the ostea, and transillumination is positive. Patient has been treated for some time locally by intranasal operations, such as middle turbinectomy and ethmoidal curettement, with but partial relief. Waiting over six months, and following the taking of this plate, we decided to do an external operation. I performed Winkler's osteoplastic operation on the right frontal and ethmoidal sinuses, breaking through the septum into the left frontal sinus, and communicating it with the right side. The Caldwell-Luc antrum operation was performed at a subsequent time, using the Vail saw to make the opening into the lateral wall of the nose. We found during the operation the anatomical and pathological condition exactly as indicated in the plate. Pathologically, the sinus contained some muco-pus and much degenerated mucoperiosteum. At another subsequent time I removed the anterior wall of the sphenoidal sinus. For the past six weeks the patient has been well.

Plate 7.—Double frontal and ethmoidal sinuitis of a chronic type, with multiple nasal polypi on both sides. No pus. Very severe frontal headache. Intranasal operation of removal of the polypi and middle turbinectomy, curettement of the ethmoid region, breaking down of the anterior wall of the sphenoid, was followed with but partial relief of the severe headaches. Six months later I performed the Hallé procedure into the frontal sinus. This is a very difficult operation and was followed by marked reaction, as there was a temperature of 104°, with very rapid pulse, and it took three weeks before these symptoms subsided and the patient recovered. But at the present time he is very well, and the nose appears to be in good condition.

Plate 8.—Multiple nasal polypi, causing complete nasal obstruction for several years. Has asthma and chronic bronchitis; marked headache over the frontal and occipital region constantly. Removed

all the polypi and middle turbinated bodies. Ethmoidal curettement. Opening of the sphenoid. Relief from the asthma and headache for a period of a few months; then the headaches recurred; in fact, increased so that an external operation was advised, but refused. You observe a cloudy appearance of these sinuses—that is, the ethmoid and frontal—which shows that the cure is but partial.

Plate 9.—Bilateral frontal sinuitis chronica. This man has been suffering from frontal headache for the past two years, almost incessantly, and uncontrollable by any method of treatment. Transillumination negative. Nasal examination: Marked deviated septum and thickening of the middle portion of the same. Sinuses show cloudy. There is no pus discharge. I did a submucous operation, as preliminary to the middle turbinectomy work. Patient very much improved since that operation. A radiograph six months later will be of inestimable value if during all that time he remains free from his headache, which then ought to show clear sinuses.

Plate 10.—Pansinuitis chronica, following a severe influenza complicated by acute pansinuitis. The symptoms are extreme dulness of intellect and pain in the head constantly, especially over the frontal region. Pus exuded from region of the sinuses; transillumination dull, antrum and frontal. This skiagraph shows the cloudiness of all the cavities. The usual intranasal treatment was of no avail. Intranasal surgery, as a middle turbinectomy and curettement, breaking down of the anterior sphenoidal wall, was followed by some relief. Three months later an external operation was advised and performed, and an osteoplastic on the frontal and ethmoidal sinuses. I used in this case the Hallé drills from above downward, also making a large opening into both antra by means of the Hallé angular drills. These procedures were followed by absolute cure. The nose is in as normal condition as one may expect after such procedures. At the time of the operation we found the condition of the sinuses corresponded exactly to the outline in the plate.

Plate 11.—Pansinuitis chronica. Has lasted four years, without any relief from treatment or intranasal operations. He has had several attacks of acute frontal sinuitis, with marked pain and some swelling externally, which, however, always subsided in a few days. Transillumination positive of the antrum, but very unsatisfactory of the frontal. External operation; Winkler's osteoplastic, etc., as in case reported in Plate 10. Recovery.

Plate 12.—Chronic pansinuitis. Local treatment did not cure suppuration. Decided to operate externally. Before so doing we

took this plate, and it showed that there were no frontal sinuses present, except two small dilatations at the root of the nose. But it does show a very cloudy ethmoid and antrum on each side. Punctures of the antra followed by washing were always associated with pus. We, therefore, did an intranasal operation, as moderately enlarging the trocar punctures, with the Myles antrum punch, curettage of the ethmoids; also enlargement of the sphenoidal sinus openings. These procedures were followed with good success. The interesting points in this plate are the absence of the frontal sinuses in both views, and it demonstrates the value of this method of diagnosis, for any procedure, whether intranasally or externally on the frontal sinus, would have very likely led to serious results.

DEDUCTIONS IN GENERAL.

1. That the real value of a skiagraph for diagnosis of sinus disease is in taking an antero-posterior exposure, as shown in Plate 1.
2. That the skiagraphs taken in the transverse view are of but very little value for diagnostic purposes, owing to the fact that one side is superimposed upon the other, but it will give an outline of the sphenoidal as well as the anterior group of cells as to their shape and size.
3. That the angle at which the tube is placed is of the greatest value, taking particular care that the rays do not have to penetrate through the massive part of the base of the skull. A transverse line seen across the orbit is found if the angle at which the plate was taken is correct, and this line is found about a half an inch below the supraorbital margin.
4. Dangerous conditions, as burn, alopecia, are possible only if one has no knowledge of the technic or carelessness. However, do not expose your patient to repeated long exposures within a brief period of time. I have never had a bad result from the use of the *x*-rays.

TRANSILLUMINATION.

The value of transillumination of the sinuses must be limited to the frontal and antra, as the ethmoid transillumination is not at all practicable. Although, as said before, skiagraphy will not prove much in posterior ethmoid and sphenoidal sinuitis. Transillumination of the frontal sinuses is of very little value, although recently Vohsen,[10] the author of this method, defends it in preference to the *x*-rays, because, he says, the latter is not practical and within the reach of very few specialists, as is his little lamp. He lays particular stress on pressing firmly the rubber protection tip of the lamp, and an absolutely dark room. It is quite different in regard to the maxillary antrum. Here the results are fairly satisfactory. The point in the technic of transillumination of the maxillary sinus is to have thorough closure of the lips when the lamp is in the mouth. The difficulties are that there is no standard light established. Abroad the small lamps are used, and in this country the large ones. I am partial to the latter, as my results are more satisfactory with the larger lamps than they were formerly with the small Vienna style.

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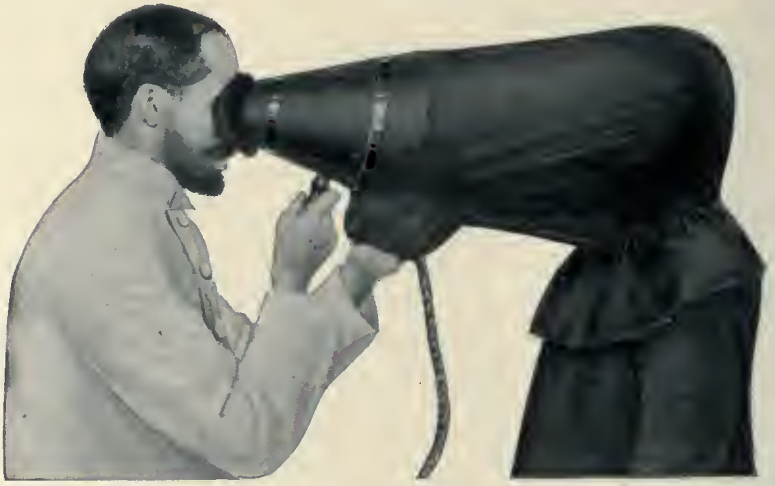
[10] Vohsen: Wiener Med. Klinik., No. 23, S. 670.



Another difficulty is in obtaining an entirely dark room. Either it is a stuffy little closet or it requires considerable time to get your office so darkened as to make the procedure practicable. I have, therefore, adopted for the past four years this method, of placing this hood over the patient, which is connected with this tube; at its end is the opening which fits very firmly about the eyes, and being held by this handle with my left hand. With the right is held the transilluminating apparatus, and passed through a slit from below which fits firmly about the wrist. Thus the openings about the examiner's eyes and about the patient's head are absolutely light-proof. I have used this method, as said before, for about four years, and am satisfied with it.

AN ORIGINAL METHOD OF OPENING THE ANTRUM OF HIGHMORE INTRANASALLY, WITH EXHIBITION OF NEW INSTRUMENTS.

Derrick T. Vail, M.D.

CINCINNATI, OHIO.

Having faithfully employed in many cases the various operations and therapeutical measures for the relief of chronic empyema of the maxillary antrum, the writer formed the following opinion nearly two years ago:

First.—That chronic antrum empyema is incurable by any and all medicinal agents employed locally in the nature of douches, either through the natural opening or through canulæ thrust through any of its walls.

Second.—That any attempt to cure by establishing drainage (?) through gold, rubber or silver tubes, whether inserted through the nasal wall, canine fossa or alveolus of a tooth, is unsurgical and only prolongs the trouble.

Third.—That the drainage and curettement of the cavity through the canine fossa with the establishment of a permanent fistula, in which a rubber plug is inserted for handy removal by the patient, who is taught to wash out his own antrum, only establishes a chronic stream of foul pus in the patient's oral cavity, is disgusting, disappointing and a failure as regards a cure.

Fourth.—That the removal of a considerable part of the turbinal wall, whether done according to the Caldwell-Luc method or any other, provided the opening is large enough, is usually successful in bringing about a cure with little or no after-treatment. But the usual method entails a preliminary operation (turbinectomy) and a period of waiting, during which time the patient is tortured with fears of the heroic operation yet to come. This feature, together with the nipping, tearing, crushing, boring procedures which are necessary with Gruenwald's and other bone forceps, in order to effect a large enough opening in the antro-meatal wall, was father to the desire to devise a simpler plan which would bring about the desired end without these objectionable features.

The grooved saw solved the problem for me. The first case had had the trocar of Krause thrust through the inferior meatus into the antrum cavity repeatedly and the cavity washed out, with no evidence that it would ever dry up. The end of the grooved saw was inserted through the opening made by the trocar and an oval window sawed out. After that there was, inside of a month, a cure which has remained permanent thus far (nearly two years).

I also learned to regard the heroic operation of Caldwell-Luc unnecessary in the cases I have managed by my method, although I do not wish to contend that the former is never indicated. The Caldwell-Luc operation has its place in surgery.

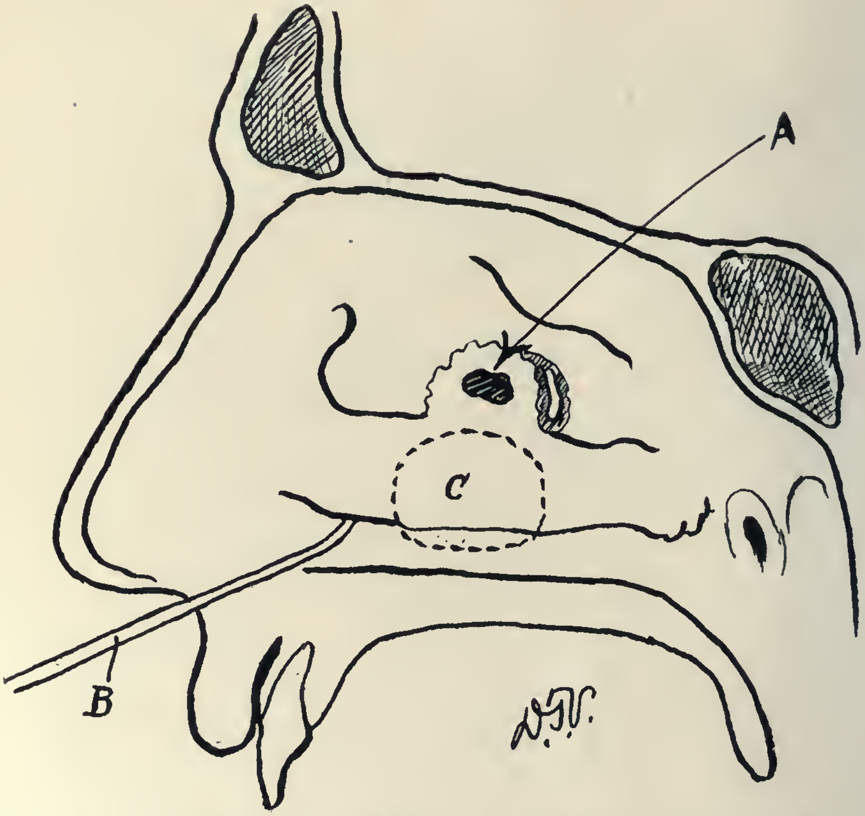


Fig. 1.—Schematic sketch, turbinal wall of the right nares. A part of the middle turbinate is removed to demonstrate the location of the ostium maxillare A., a probe, B, is represented as pointing to the outlet of the nasal duct. The dotted oval line, C, represents the size and location of the button of bone it is proposed to remove by means of Vail's grooved saw, making a window opening into the antrum.

I have operated on seven successive cases of chronic maxillary empyema with the grooved saw with such satisfactory results that I would not subject a case to any more severe operation, at least until this method which I am about to describe had been used and proven inadequate.

The grooved saw, made for me by Max Woeher & Son Co. of Cincinnati, can saw in only one direction, and that is circular or oval. The groove is carefully constructed to shallow depth, which will not permit a small opening to be made, for it is necessary to remove a large button of bone if the end of the operation is not to be frustrated.

The principles involved are the following: (a) free drainage of a foul cavity; (b) ready accessibility to a naturally inaccessible bone cavity; (c) establishing a permanent opening in a reservoir of pus to which there is but one natural opening, thereby making another large opening near the floor of the cavity, as it is well known that a reservoir or receptacle having only one opening can not readily empty itself. This principle is easily proven by drawing off a fluid, say vinegar, from a barrel where there is always a large opening, the faucet, for draining off the fluid and a small opening, the vent, for admitting the air; (d) establishing free ventilation in a foul chamber which normally contains but one natural window. This principle is employed in our sleeping and living rooms when we wish to ventilate. It is always necessary to have two openings, at least, if we want fresh air. (e) Establishing an opening in the pathway of a stream of antiseptic fluid ejected from an ordinary douche, thereby enabling the patient to wash out his own antrum by simply douching that side of his nose, should it be required. (f) The establishment of an opening that will permit of inspection, packing and medication.

The advantages of this method are:

The painless operation under local anesthesia; the lessening of personal risk to the patient; the speedy execution of the entire operation (in one case the operation was completed in three minutes, in another five minutes; ten minutes is the usual time); the operation completed without the necessity of a preliminary turbinotomy and the cure resulting without the wearing of plugs and plumbing devices.

Instruments Used.—Kramer's speculum, angular packing forceps, flexible copper probe, Krause's probe, angular scissors that will cut well at the end, special hypodermic for the cocain and adrenalin mixture, special perforating bistoury (Vail's), special grooved antral saw (Vail's).

The Operation.—Having sterilized the patient's nose, lip and nasal vestibule with soap and water and bichlorid, douched the nasal cavity and front and rear by postnasal douche, the hairs in the vestibule are cut short and pledgets of cotton soaked in 5 per cent. cocain are packed under, upon and above the inferior turbinated bone. A wait of seven minutes by the watch is allowed, during which time the operator can busy himself by seeing that the special aseptic glass syringe is properly sterilized and loaded with equal parts of 2 per cent. cocain and 1/2000 adnephrin. This will reduce the cocain to 1 per cent. solution and the adnephrin to 1/4000. The cotton is withdrawn from the nose and the operative field brushed with 1/5000 adnephrin solution, then the needle of the hypodermic is inserted under the mucous membrane of the turbinal in the site of the operation, which is, be it remembered, not the front end, but the middle third of the turbinate. The solution is driven under the mucous membrane on the lateral wall *beneath* the inferior turbinate, as well as on the convexity of it. In all about twenty drops are injected. As soon as the needle is withdrawn the actual operation begins, there being no need of further waiting. The perforator is passed in the nose, point downward toward the floor. When the middle of the inferior turbinal is reached, it is turned directly toward the antrum and thrust through the bony partition under the inferior turbinate by a positive pressure, at the same time forcing the handle of the instrument toward the septal side. When the antrum is entered, the instrument is forced backward toward the pharynx and forward toward the nasal entrance to enlarge it in order to insure entrance for the saw, which is next used. The saw is inserted well through the slit with the teeth toward the antrum cavity. The tip of the nose is deflected toward the other side by the nasal speculum to permit inspection and a free sweep. The timid operator who lacks the surgical instinct will fail at this point, unless he makes up his mind that there is nothing to fear. The saw is grasped firmly, the patient's head steadied by an assistant, and a few bold strokes of the saw, keeping in mind the intent of making

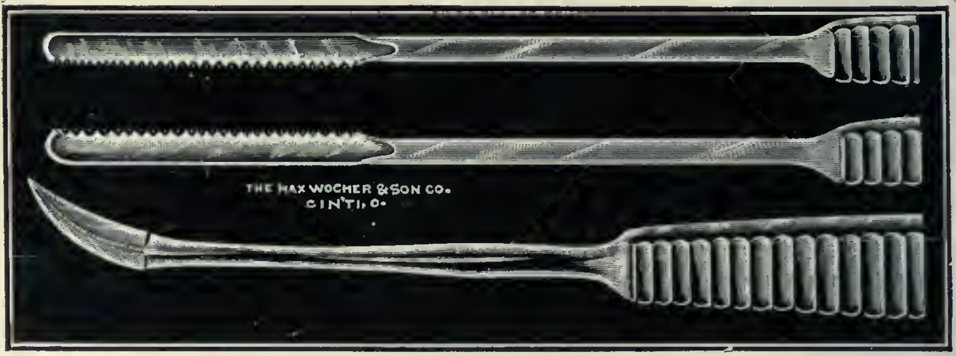


Figure 2.

an oval opening and causing the saw blade to describe the oval in its course, which it naturally does, coming out where it first started, will render the maneuver successful, even in the cases of narrow nares. There is usually an overhanging apron of mucous membrane which the saw, in its oblique course through the posterior half of the oval, has undercut. This is easily cut away by angular scissors or Gruenwald's forceps. The cavity is now open for exploration with the large probe of Krause. A little aristol powder insufflated

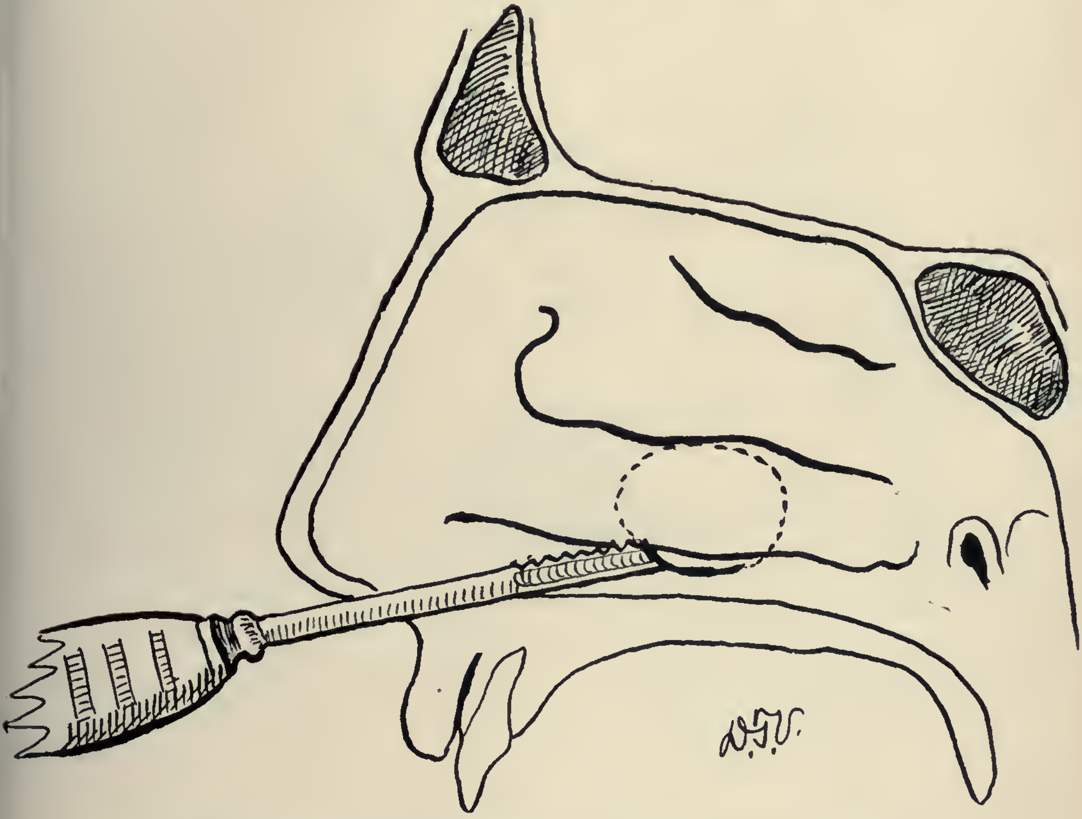


Fig. 3.—A slit opening has been made in the antrum wall under the inferior turbinate by means of Vail's perforator. The end of the grooved antrum saw has been passed through the slit and the dotted line shows the route proposed to be taken by the saw in its natural course.

will aid the vision in locating the opening. The cavity should be washed out with sterile salt solution and packed with a moist gauze strip, which is easy to do, and the operation is completed. The patient is ordered to wear a cotton plug in the nostril for three or four days to guard against outside infection.

Should the cavity be filled with polypi and granulations, a condition I have not encountered among the cases cured by this method, it can be partly cleared out by means of curettes; but not entirely so, for the front half, at least, is out of the field of curettement. In that case I would not hesitate to inject the cocain-adnephrin mixture under the mucous membrane of the canine fossa, expose the bone by an incision one inch long, peel back the periosteum, perforate the front wall of the antrum by means of a short, strong knife and saw out a circular or oval button of bone with the same grooved saw that was used in the intranasal operation. This method would expose the antrum to thorough inspection and would, in fact, be a Caldwell-Luc operation with technic reversed. I have done the Kuster operation through the canine fossa under local anesthesia and it was painless. Pynchon of Chicago has preceded me in the invention of a grooved saw.[1] Pynchon calls his a saw curved on the

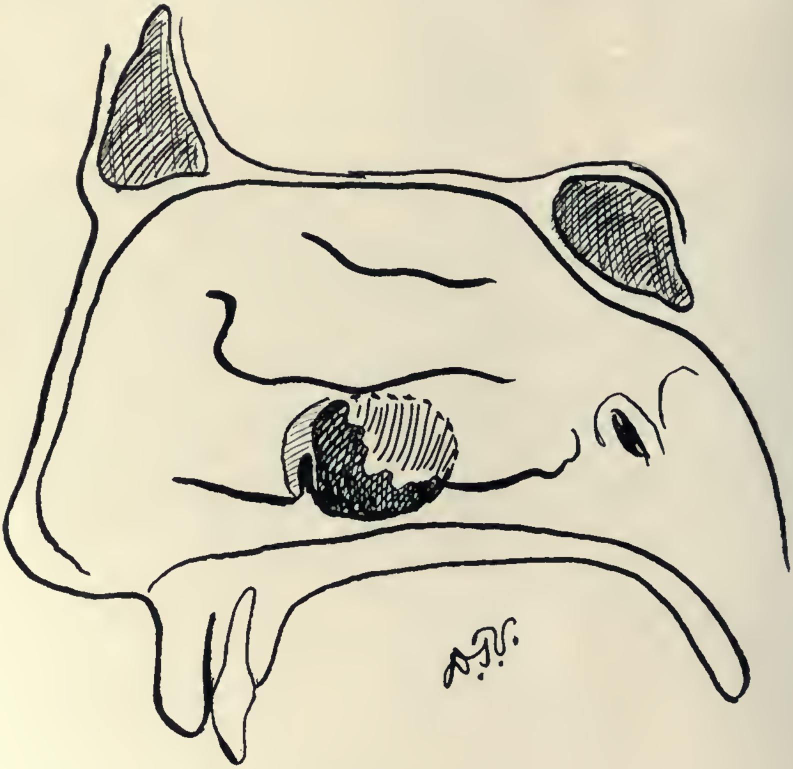


Fig. 4.—The button of bone has been removed, but since the sawing was done at an oblique angle with the plane of the turbinal wall there is usually a flap of mucous membrane which overhangs the opening, this flap should be cut away.

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[1] Annals of Otology, Rhinology and Laryngology, February, 1900.

flat, and it is of different shape and designed for low spurs. His saw, which is doubtless of great value for the purpose for which it was designed, would not do the work which my saw does in this operation.

The antral saw I have devised for the operation has a rather wide blade and a shallow groove. The teeth are sharp and should be kept so to work well. It will saw in a curved pathway and return to its starting point.

It will be observed that in this operation it is unnecessary to do a preliminary turbinotomy. The saw will leave the useful front end of the inferior turbinate and take only that part which is necessary to accomplish the success desired. In this way the valuable erectile tissue on the front end of the turbinate is conserved, a thing greatly to be desired, for, be it remembered, that the physiological action of this tissue has the analogous action in controlling the entrance of air of varying humidity and barometric pressure in the nose that the iris has of controlling the amount of light intensities which enter

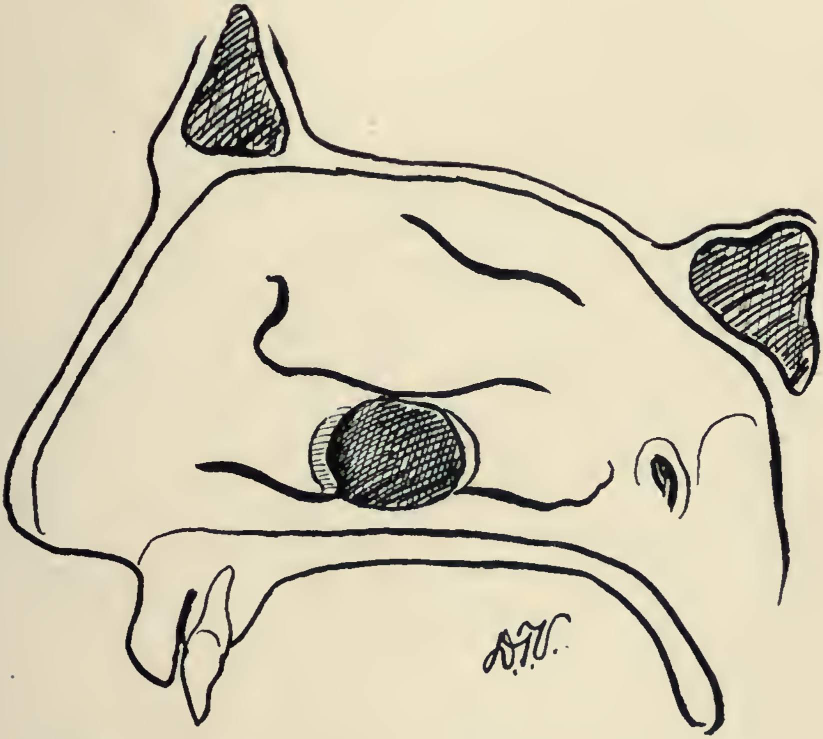

Fig. 5.—Showing the relative size of the opening, its location and appearance at the close of the operation.

the eye. Something has been said about the antrum opening which is made by the intranasal operation not being at the lowest point of the antrum cavity. It is true the antrum cavity dips considerably lower when the floor of the nose is horizontal, as in the upright position of the head, but it is such an easy thing to tilt the head to one side and make the nasal opening the lowest point that I can see no force to that argument. During the sleeping hours, while the patient lies on the opposite side, the cavity would certainly drain out.

I have repeatedly demonstrated a point raised in the premise of this paper, namely, that the current of water from the douche will find free entrance to the antrum after the operation, for after such an anterior douching of the nose and the fluid has been blown out, the patient will empty a spoonful more by tilting his head, proving the point beyond question.

The sign of a cure is when there is no longer any catarrhal discharge and the nose is clear and free. The continuation of the douche after this point has been reached is to be deprecated, for there is nothing gained by douching a healthy cavity. Moreover, the patient will sooner or later infect his cavity with foul water through carelessness, not to speak of the other dangers of the douche habit. When the cavity is well, the patient should be instructed to stop all treatment.

A PLEA FOR CONSERVATISM IN THE TREATMENT OF CHRONIC EMPYEMA OF THE MAXILLARY SINUS.

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There has been so much said and written on chronic empyema of the maxillary sinus during the past year that it seems like "carrying coals to Newcastle" to try to add anything. Nearly all the recent literature to which I have had access, with noticeable unanimity, agreed upon the intranasal opening and treatment of this disease, and has decried the so-called Caldwell-Luc operation (which is usually accepted as the radical one) save in exceptional cases. To all of this I am in accord; but each writer has, it seems to me, vied with his confrère in destroying as much as possible of the nasal wall of this cavity, and it is remarkable the ingenuity displayed in devising new chisels, saws, drills, etc., for this purpose, each possessing some special advantage in the hands of its inventor according to his special methods and skill.

Historically, it is interesting to trace the various ways and means of treating this malady since Nathaniel Highmore, in 1651, described the sinus. Following William Cowper with his operation that still bears his name (although he probably was not the first to open the antrum through the socket of a tooth) came Gooch and John Hunter, who independently proposed an opening through the nasal cavity, and a Swedish surgeon with an unpronounceable name who operated after the manner of Cowper and then made a counter-opening into the nose through which he passed a second canula, and so on down to the paper of Ziem, in 1886, whose essay was the first to describe the frequent occurrence of this affection without the classical symptoms of pain, swelling and tenderness, and which was the beginning of our present methods. This is so striking when compared with the writings of to-day that Jonathan Wright, from whose book most of my historical references are taken, says: "I am sure any one, reading these accounts of operations on the antrum of Highmore for suppurative disease, will perceive that all the recent procedures, which have been of late so exhaustively and frequently described, have been long anticipated in surgery."

Our critics have said, "We move in a circle," to which our friends reply, "that it is a spiral and each revolution finds us on a higher plane."

I am not here to-day with any new methods or appliances, but to cite a few cases treated by opening the antrum through the inferior meatus after the manner proposed by Krause in 1889, and washing the cavity daily with normal salt solution. These canulæ are from four to five millimeters in diameter, permitting a large volume of solution to enter and sufficient pressure to cleanse the cavity, being one of the numerous advantages over treatment through the natural opening or the use of a small canula. That this method is not applicable to cases where polypi or other tumors exist is self-evident, but that a number of cases are cured without the unnecessary destruction of the nasal wall and leaving a permanent unprotected opening into the antrum is, I think, proven. It is too early to claim that this entrance into the cavity never does harm; certainly it does no good.

My technic is to make the puncture under the inferior turbinate, as close to it as possible, from 1.5 to 2 cm. posterior to its anterior attachment, then washing the cavity thoroughly with the bulb of a Holmes' or Vail's nasal douche attached to the canula, using, as stated above, the normal salt solution. I find the bulb much better than a syringe, as the pressure can be controlled and sufficient force exerted to remove all inspissated pus or other débris equally as thoroughly and with much less damage to the mucous membrane than can be done with a curette; at the same time the natural opening is cleansed of scabs or dried secretion from this *vis a tergo,* which, if Halle is correct, permits the cavity to be cleansed by suction from the inspired air. These washings are continued daily until the solution is free from pus, then every other day for two or three times, and then after one week, and if there is no pus the patient is dismissed as cured. This must be modified to suit the requirements of each case.

Case 1.—Patient came to me Nov. 19, 1905, with a history of having had pus in the right frontal sinus, for which he had been treated for six months through the nose without benefit. Pus flows from right nostril all the time and reappears in the region of the hiatus semilunaris when wiped away. A probe readily entered the frontal sinus.

The Krause trocar with canula was easily introduced through the inferior meatus, the withdrawal of the trocar being followed by some foul-smelling pus. The cavity was thoroughly washed until the salt solution came away clear; again the next day this procedure was repeated, there being a large amount of pus; this was continued every other day for ten days, when, to my surprise, the washings were clear and free from pus. I was informed a few months ago that the patient had not had any further trouble and regarded himself as permanently cured.

Case 2.—Mrs. B., aged 53, came to me Aug. 23, 1905, with loss of sense of smell and a constant purulent discharge from the right nostril, which patient stated had continued since an attack of septicemia fifteen years ago. Both middle turbinates were large and pressing against the septum, right nostril being much smaller than the left. I removed the middle turbinates. On November 18 I removed the right inferior turbinate preliminary to doing a radical operation on the maxillary antrum. One month later the patient, who had been a semi-invalid for years, begged me to try some expedient that would relieve her and not tax her strength like an operation under general anesthetic. I made an opening in the nasal wall with the Krause trocar and canula and washed the cavity, which was filled with thick pus, thoroughly. This washing was kept up daily for a long time; then every other day, and finally once a week until May, when I discharged the patient as cured. To-day she remains free from the constant dull headache, her general health is much improved and the secretion in her nostrils normal.

Case 3.—Female, aged 35, came complaining with headache and a constant dripping in the throat and a disagreeable odor that nothing seemed to remove. Found both middle turbinates enlarged and pressing against septum, some pus in left nostril in region of middle turbinate. Removed anterior ends of both middle turbinates Jan. 14, 1907; on April 12 opened left maxillary antrum with the Krause trocar and canula, enlarging opening slightly with Myles' back-cutting chisel, finding considerable pus of foul odor; continued washing cavity until April 20, when washings were free from pus and patient was dismissed as cured. This patient thinks that this trouble began three years before with an abscess at root of first molar tooth.

Case 4.—Mrs. D., aged 50, came to me in April, 1907, with a history of antrum trouble first diagnosed by her dentist several years ago; was complaining of being annoyed by a constant foul odor, worse after stooping. Had just been examined by her dentist and family physician, who sent her to me for treatment of antrum disease. I found the nostril clean and practically normal; transillumination showed the face very clear, but left pupil could not be seen. I was much inclined to doubt the correctness of this diagnosis, but decided to make an opening through the inferior meatus, which I did with Myles' chisel, and found foul-smelling odor. This cavity was washed daily at first and then less frequently until the middle of May, when patient was discharged, the washings from cavity being free from pus. Patient returned in August, stating that her dentist claimed there was pus which he had been able to reach through a cavity in the third molar tooth, stating that he was able to introduce a probe through the tooth into the antrum. This I was unable to do, and, as the pupil was perfectly illumined by the usual method and she had none of her old symptoms, I sent her back to have the decayed tooth extracted, assuring her that would end her trouble. I have not heard from her since.

Case 5.—Patient has had a purulent rhinitis with headache for many years, the septum has a very large perforation, no illumination of pupil or region of maxillary antrum, has more pain and pus in left nostril. Opening through inferior meatus showed much pus in antrum not fetid. Patient lives seventy miles away, and has not been treated with any regularity, but is much improved and free from her distressing symptoms of heaviness and pain. No bacteriological examination was made of discharge.

Case 6.—This patient has been a chronic sufferer from neuralgia for years. She came to the office with an acute exacerbation much worse, as are all of her attacks, on the left side. Examination revealed pus in left nostril, which flowed freely after cocainizing the middle turbinate and gave relief from pain. Made an opening through the inferior meatus with Myles' chisel, finding pus; washed cavity thoroughly and patient had a restful night, free from pain. In two weeks patient went to the country, having had no pain since first treated, and at the time of departure no pus in the cavity, as demonstrated by douching it.

Case 7.—Male, aged 38, does not know when empyema of maxillary antrum developed, or the cause. The cavity was opened through the socket of the second bicuspid tooth by my predecessor, who gave up practice over ten years ago. At present there is a disagreeable odor of which the patient is conscious all the time and his associates frequently. There is a fistula at site of old opening which he keeps patulous with a toothpick, or anything convenient; after dilating this canal he is able to force fluid from his mouth into the antrum and has been cleansing it in this manner for years. Opening and washing through inferior meatus brought pus in abundance and fetid odor. Since the first washing the odor has subsided and very little pus; after six weeks' treatment patient was dismissed as cured.

I think that from these few cases, selected for the reason that they were unquestionably chronic, we are justified in trying this simple procedure at least for a few weeks before destroying the nasoantral wall, that we are safe in concluding that the mucous membrane only needs thorough cleansing in a large number of cases to render it free from disease, and that washing it thoroughly can accomplish this as effectually and with much less destruction than the curette.

THE VICIOUS CIRCLE OF THE NOSE.

THE RATIONALE OF SINUS INFLAMMATIONS AND THEIR TREATMENT.

William Lincoln Ballenger, M.D.

CHICAGO, ILL.

A somewhat extended clinical experience, and a knowledge of the anatomical arrangement of the structures of the nose, together with a knowledge of well-known laws regulating the predisposition of mucous-lined cavities to infection and inflammation, have led me to hold the opinion that a large percentage of the cases of sinuitis affecting the sinuses draining into the middle meatus of the nose, may be successfully treated by intranasal operative procedures. I am likewise convinced that only in exceptional cases is it necessary to perform extranasal operations for the cure of infection and inflammation of this group of sinuses.

When we recall the fact that the frontal and maxillary sinuses are included in this group of sinuses, and that divers external operations have been advocated and practiced for the relief of infections and inflammations affecting them, my claims for intranasal surgery may at first be thought to appear too strongly stated. I trust, however, in this paper, to state the facts, as they have occurred in my practice, in such a way as to substantiate the promises herein stated.

The Vicious Circle of the Nose.—The frontal, anterior ethmoidal and the maxillary sinuses have a common exit for the discharge of their normal and diseased secretions, namely, the infundibulum. While this statement is open to certain exceptions, to be noted, it is, nevertheless, in the main, true. The frontal sinus does not uniformly drain into the infundibulum, but, according to Logan Turner, it drains directly into the middle meatus of the nose in 50 per cent. of the skulls examined by him. Nor do all the anterior ethmoidal cells drain into the infundibulum. The bulla ethmoidalis and middle turbinal cells are exceptions. While the osteum maxillare always opens into the infundibulum, it by no means constitutes the only source of drainage for the antrum in a large number of subjects. Turner found accessory maxillary ostei in four of the nine specimens examined by him, the accessory ostei being posterior and a little inferior to the posterior end of the hiatus semilunaris. With these exceptions the infundibulum and its osteum (the hiatus semilunaris) receive the secretions from the frontal, anterior ethmoidal and maxillary sinuses.

This being true, it is obvious that any condition, anatomical or pathological, causing obstruction to the flow of the secretions from the infundibulum via the hiatus semilunaris will interfere with the drainage and ventilation of one or all of this group of sinuses.

The well-known law referring to the predisposition of mucous-lined cavities to infection and inflammation may be stated as follows:

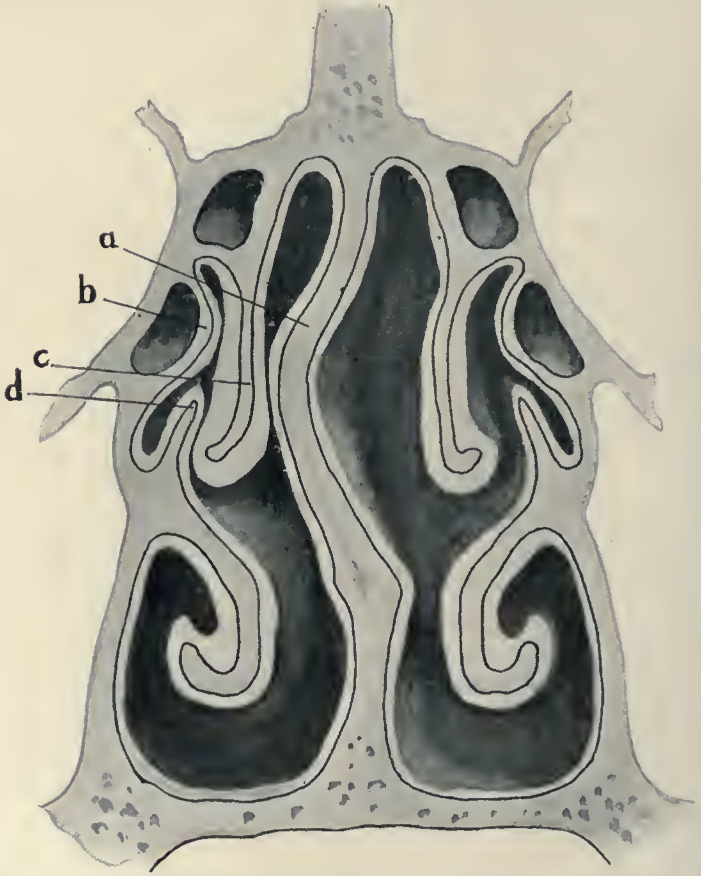

Fig. 1.—a, Closure of the infundibulum by a deviated septum; b, the inner wall of the bulla ethmoidalis; c. middle turbinate; d, uncinate process or inner wall of the infundibulum.

Any cavity lined with mucous membrane is predisposed to infection and inflammation when its drainage and ventilation are impaired. In view of this law it is obvious, therefore, that any condition, anatomical or pathological, causing obstruction to the flow of the secretions from the infundibulum, via the hiatus semilunaris, will predispose the mucous membrane lining the sinuses draining into it, to infection and inflammation.

It is equally obvious that if the obstruction to the drainage and ventilation is removed the predisposition to infection and inflammation will disappear. That is, whereas, the obstruction to the drainage and ventilation causes a lowered resistance of the tissues, the removal of the obstruction raises the resistance of the tissues, hence the disappearance of the infection and inflammation.

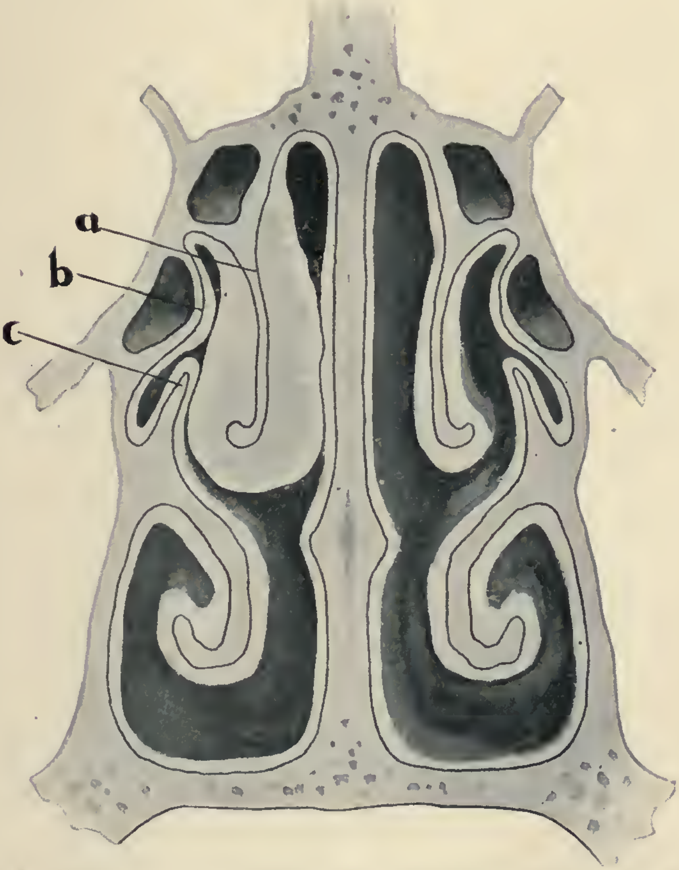

Fig. 2.—Closure of the infundibulum by an edematous middle turbinate (a).

The clinician may, with truth, say that it is a condition that confronts him, and not a theory. The question, therefore, resolves itself into the query, Does a sinuitis affecting this group of cells tend to disappear when obstructive lesions are removed from the region of the infundibulum? According to my experience it does, though there are important exceptions.

Having outlined the hypothetical aspect of the problem, it remains to present concrete data as to the actual results obtained by procedures instituted in consonance with the hypothetical statement. In order to make the clinical deductions more clear, additional anatomical data will first be given.

(a) The nasal septum is frequently deviated toward the lateral wall of the nose in the region of the anterior half of the middle turbinated body, often crowding the middle turbinal against the outer wall of the nose (Fig. 1), thus obstructing the drainage and ventilation of the frontal, anterior ethmoidal and maxillary sinuses.

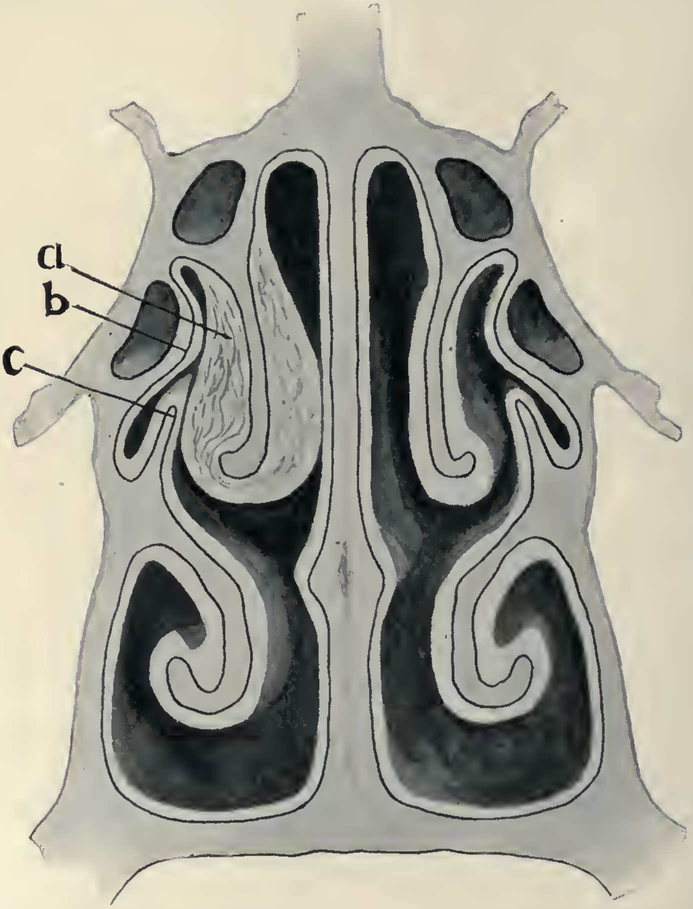

Fig. 3.—Closure of the infundibulum by an hyperplastic middle turbinate (a).

(b) The middle turbinated body is frequently enlarged by edema (Fig. 2), hyperplasia (Fig. 3) or by the presence of accessory ethmoidal cells in its body (Fig. 4) and may in consequence block the hiatus semilunaris; or the middle turbinal may cling so closely to the outer wall of the nose (Fig. 5) as to block the hiatus.

(c) The bulla ethmoidalis is located immediately above the hiatus, and when enlarged it may overhang the hiatus and completely obstruct it (Fig. 6).

(d) The lip of the uncinate process, or median wall of the infundibulum, may be the seat of accessory pneumatic cells which may obstruct the infundibulum.

The anatomical structures described above I am pleased to call the "Vicious Circle of the Nose" (Fig. 7).

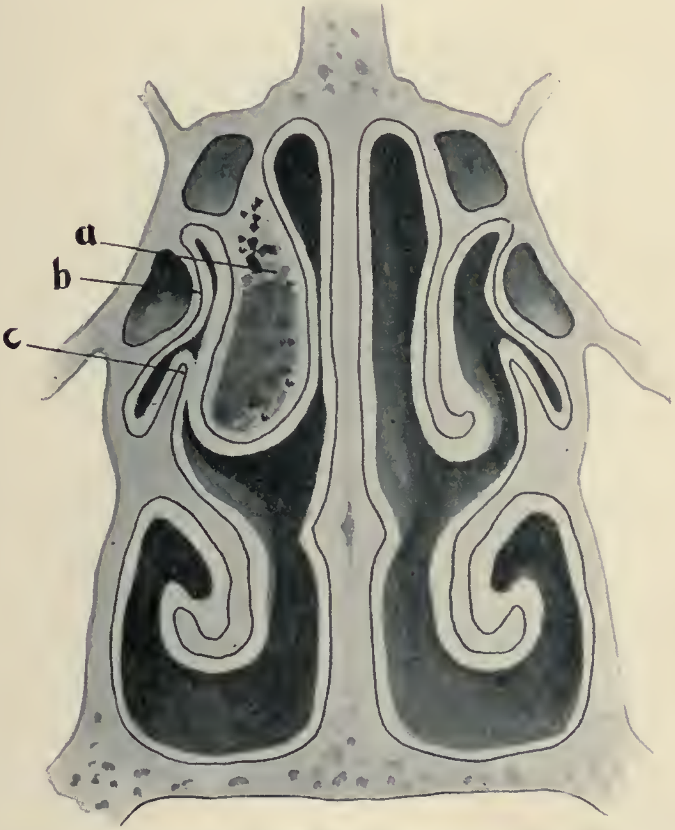

Fig. 4.—Closure of the infundibulum by a cystic middle turbinate (a).

I do not use this expression because I wish to introduce a new terminology, but because I hope by it to emphasize the clinical importance of the various anatomical structures embraced within its limits. I shall hereafter, in this paper, refer to the anatomical structures within this area as the "vicious circle" of the nose, because it is in this area, rather than in the sinuses, that we must look for the predisposing causes of the infection and inflammation. Finding the cause here, we should address our remedial measures to its removal; should these efforts fail, more radical measures may be undertaken.

Clinical Data.—From an anatomical view it has been shown that in simple sinuitis of the frontal, anterior ethmoidal and the maxillary sinuses, all of which usually drain into the infundibulum, the obstruction to ventilation and drainage is often due to the malposition or diseased condition of the structures in the vicinity of the infundibulum, rather than in the sinuses. This being true, we should expect the removal of the obstruction in this region, the "vicious circle," to be followed by the relief or cure of the sinuitis. If the obstruction were located in the sinuses, or at their ostei, the removal of the structures of the "vicious circle" would not be followed by a relief or a cure of the sinuitis. Under such conditions

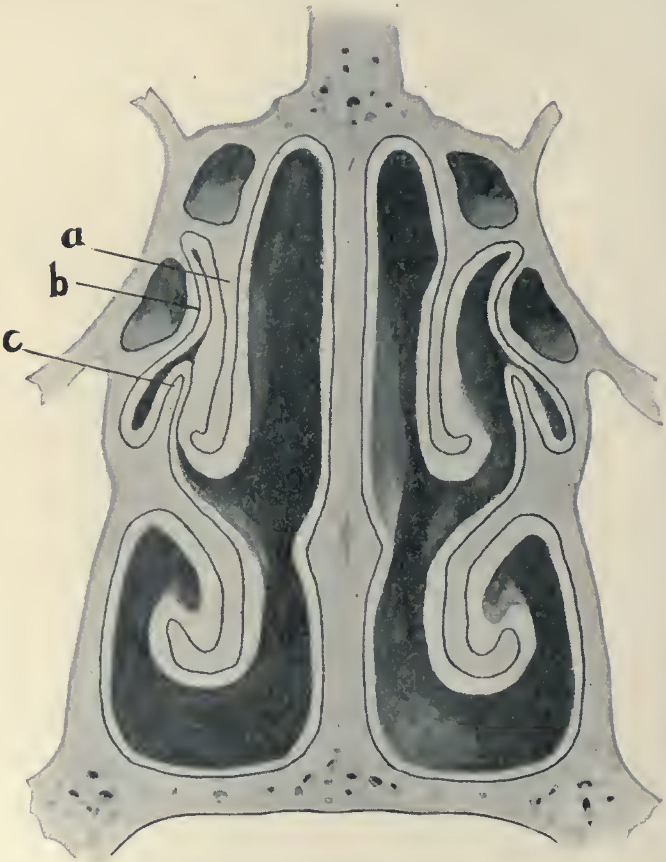

Fig. 5.—Closure of the infundibulum by a closely hanging middle turbinate (a).

it would be necessary to direct the therapeutic measures to the sinuses or to their ostei. My clinical observations cover some two hundred operative cases for chronic sinuitis affecting the frontal and anterior ethmoidal sinuses, and in but five instances have I found it necessary to extend my operation beyond the "vicious circle" of the nose. The diagnoses in seventy-five of these cases were confirmed by skiagraphs, and in no instance was the skiagraphic finding at variance with the diagnosis previously made. I infer, therefore, that in the earlier one hundred and twenty-five cases my diagnoses were probably correct.

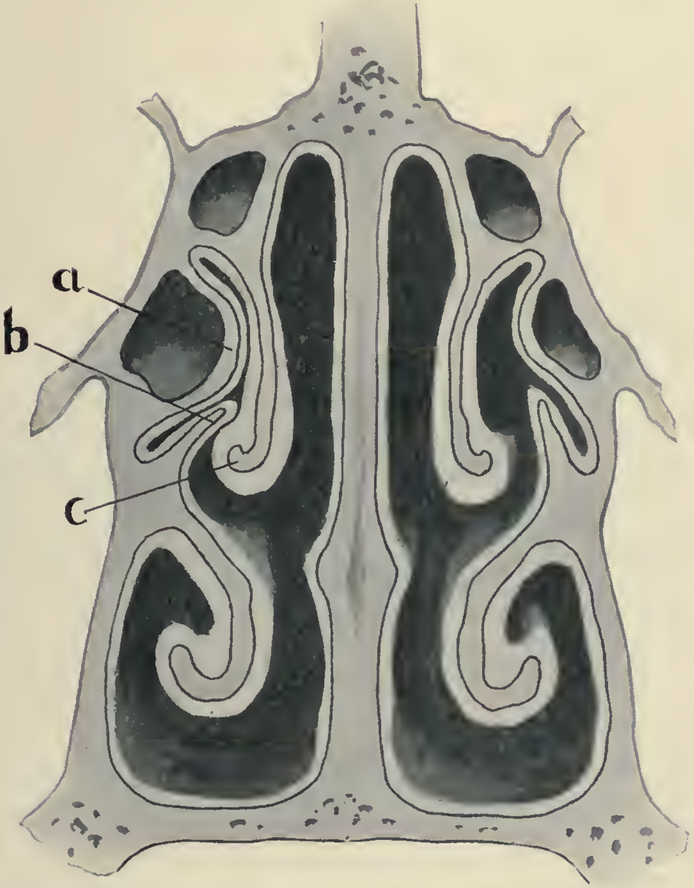

Fig. 6.—Closure of the infundibulum by an enlarged bulla-ethmoidalis (a) impinging against the uncinate process (b).

In view of these observations I may fairly conclude that a very large majority of the cases of inflammation of the frontal and anterior ethmoidal sinuses may be cured by limiting the treatment to the area of the "vicious circle" of the nose. While the percentage of cases thus apparently cured by me is 97.5, I do not wish to go on record as claiming this to be a correct estimate of the effects of this mode of treatment. There are several reasons to be offered against the accuracy of such a conclusion.

In the first place, the number of cases (200) observed by me is too small to warrant an accurate statistical statement.

In the second place, the question of diagnosis must be taken into account. Many observers only diagnosticate sinuitis when the symptoms are very obvious, the presence of pus in the middle meatus of the nose being the objective symptom most often depended upon. If only such cases are to be regarded as sinuitis, it is obvious that a much smaller percentage would be cured by directing the therapeutic measures to the "vicious circle" of the nose. In some of these cases the disease is much further advanced, and the tissue changes more pronounced in the sinuses and around their ostei; hence, in addition to the blockage of the infundibulum, the blockage from the granulations, etc., in the sinuses and around

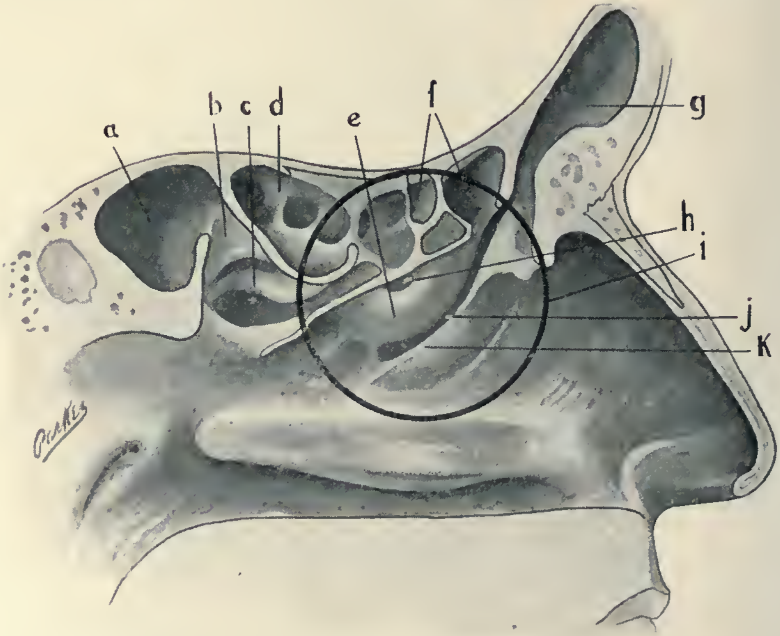

Fig. 7.—The vicious circle of the nose. The key to the sinus diseases: (a) the sphenoidal sinus; (b) the spheno-ethmoid space; (c) the superior turbinate; (d) posterior ethmoidal cells; (e) the bulla-ethmoidalis; (f) the anterior ethmoidal cells; (g) frontal sinus; (h) osteum bulla-ethmoidalis; (i) the circumference of the vicious circle; (j) the hiatus semilunaris or slit-like osteum of the infundibulum; (k) the uncinate process or the inner wall of the infundibulum.

their ostei must be taken into account in carrying out the therapeutic measures. Bone necrosis and intracranial complications also call for the radical surgical treatment. All cases with obvious purulent discharge into the middle meatus of the nose do not, however, come under the complicated class, many of them being amenable to treatment directed to the structures of the "vicious circle." As previously stated, only five out of two hundred cases coming under my care required radical operative procedures, and in all of these I performed the Killian operation, four with brilliant results both as to a cure and to external deformity, and one with an entirely negative result.

In my diagnosis I have included some cases in which I could not see pus, though the patient claimed he frequently blew it from the nose, especially early in the morning. They complained of frontal headache, either unilateral or bilateral, usually more pronounced of mornings. They also complained of dizziness of short duration. which was aggravated upon stooping. The anterior end of the middle turbinated body was often enlarged, glazed, velvety in texture and rested against the septum. In some instances the septum was bowed in the region of the middle turbinal and forced the turbinal against the outer wall of the nose, and thus prevented free ventilation and drainage of the sinuses draining into the infundibulum. In others the bulla ethmoidalis was enlarged and blocked the hiatus semilunaris. These findings, and the cloudiness of the frontal and ethmoidal cells upon the skiagraphic plate, led me to make the diagnosis of frontal and anterior ethmoidal sinuitis.

In cases thus diagnosed I believe that 90 per cent. of them may be cured by addressing the treatment to the "vicious circle" of the nose, while the remaining 10 per cent. will require external operative procedures. Of the cases curable by limiting the treatment to the "vicious circle" of the nose many are curable by non-surgical methods, as the local applications of adrenalin, cocain, a 10 per cent. glycerin solution of ichthyol, antipyrin, etc. Still others may be greatly benefited by divulsing the middle turbinal away from the septum, or away from the outer wall of the nose, according to its point of impingement or obstruction. In the remaining cases it will be necessary to either remove the anterior half of the middle turbinal, break down the enlarged and overhanging bulla ethmoidalis, or even to exenterate the anterior ethmoidal cells as thoroughly as possible, and in rare instances to remove the floor of the frontal sinus by Hallé's method. In those cases complicated by inflammation of the posterior ethmoidal and sphenoidal sinuses the surgical treatment should be extended to include them in its scope.

In conclusion, I wish to say that the object of this paper is to call attention to the fact that the obstructive lesion which interferes with the ventilation and drainage of the frontal, anterior ethmoidal and maxillary sinuses is usually not located in the sinuses, or even in their ostei, but is in the hiatus semilunaris and infundibulum or in the structures in their immediate vicinity. This being true the rational therapeutic indication is to remove the obstructive lesions from this region, the "vicious circle" of the nose, rather than to attack the sinuses themselves. In the smaller percentage of cases in which extensive tissue change has taken place, a radical external operation should be performed.

DISCUSSION ON PAPERS OF DRS. ANDREWS, BECK, VAIL, MOORE AND BALLENGER.

Dr. Minney:—I have been a sufferer from antral trouble, and I commend the method pursued by Dr. Moore. I think it is a good method after internal medication and sprays have been used. The internist has his place in these conditions. We must not forget that topical applications can be used to advantage, and they should be tried first. The second plan would be that pursued by Dr. Moore. I prefer that to the other, if that will do the work. Dr. Vail spoke of the drainage, and by having two holes there you could get better drainage. I am sure the doctor will not be offended when I say I think he made a mistake. Those cavities, where there is but one opening, are freed by suction, and particularly in the antra we find such is the case. When you make two openings, you do not have suction any more, and you must depend upon flushing. So if it is possible, we want to use that as a last resort. I am well pleased with Dr. Vail's suggestion and plan of treatment, but let it be the last resort. I was in a city not very long ago in which they were doing turbinectomies on a wholesale scale, and they were removing every enlarged one, seemingly. That is a vicious practice. It is all right to remove the turbinate if it is necessary, but we are liable to become too aggressive. So I say, if Dr. Moore's method will do the work, let it be done. In my own case, Dr. Brown opened up the antrum through the alveolar process and put in a tube there that annoyed me very much, washed the antral cavity a few times, and then told me to continue flushing. The tube was painful (I am not speaking disrespectfully of Dr. Brown), but in a very short time, when there was no odor and no detritus, I let it alone and it got well. And in those cases, as has been suggested, if we will establish drainage, that is the essential point. Three months ago a lady came to me for some trouble, could not breathe through the left nostril. She had a little patch on the eyelid. She said she had a running sore. She had a fistulous opening from the frontal sinus. The nose was filled with polypoid masses. My partner suggested an opening of the frontal sinus, but we removed the polypoid mass, and in three days there was nothing but a little moisture from this opening, and in ten days the fistulous opening into the frontal sinus was closed. It remained closed until she thought she was well—probably some two weeks—when she went visiting, and when she came back there were some more polypoid masses in the nose. The nose was plugged up, and that sinus was open again, illustrating Dr. Ballenger's position beautifully. But what I want to get at is, let us avoid these openings if possible; but if there is no other way, then let it be done. I am satisfied that free drainage through the nose would cure the frontal sinus case alluded to.

Dr. McCaw:—The method which Dr. Moore uses is simply a method which he has adopted for use in his cases, and not his own. The value of any particular method of opening into the antrum is to get drainage. Now you get drainage from the nasal cavities every time one clears the nostril. At such times they blow out any obstruction that is there. That, in a measure, helps to keep the antral cavity empty where there is an opening in the nasoantral wall. Another important point is to use force in flushing out the antral cavity. I prefer a syringe with Killian's tip for this purpose. After flushing the cavity, it is my custom to clear out the retained fluid by forcing air from an empty syringe through the rubber tube and canula used in the flushing; in this way the antral cavity is left quite dry and free of secretions. Dr. Vail, I think, objected to flushing the antrum on account of the possibility of retained fluids. It seems to me that by following out the above method of flushing this objection is overcome and the cavity left in a very much cleaner condition.

Dr. Brawley:—As Dr. Andrews suggested, it may be interesting in this connection to show an apparatus, which I have not invented by any means, but have simply arranged for the purpose of bringing about a vacuum in the nasal cavity. It is a chemist's pump, which is arranged for me by the W. R. Grady Co. of Chicago. It is used commonly as a filter pump. This is attached to an ordinary water tap, with a handle which is adjustable, so that you may turn on as much water as you choose, thus regulating the suction force. Water running from this tap past a slot in this attachment abstracts the air from the tube. Here we have a bottle that collects any secretion coming over from these tips which are to be introduced into each nostril. In using this apparatus I am in the habit of cocainizing the nose and using suprarenalin almost as freely as if I were going to operate, so that I secure every bit of contraction of the nasal tissues possible. Then, "putting them on the pump," as I often say, we introduce one of the glass tips into the nostril, and the patient is instructed, by one of several different methods, to raise his soft palate—such as saying "hic"—or starting to say the letter "k"—or, as we frequently hear—"scraping the throat." Or, if he opens his mouth very widely, as though he were going to gag, he may get this position much easier. It requires half a dozen different suggestions to get the throat in the right position. As a rule, the patient makes too great an effort, as he thinks it a reflection on his mentality not to be able to control his throat, but I have had only one man whom I could not teach, and that was a man who was spending most of his time in a sanitarium. I have one particular case where I used the suction in diagnosis. I had been washing the cavity for two or three months. I could not reach the sphenoidal ostium by operation—it was denied in this case—and after I had arranged this apparatus it took me about three minutes to make the diagnosis. You could see the pus running down over the posterior end of the middle turbinal, and even over against the septum posteriorly. I usually leave the suction on for about five minutes. The operator can tell when suction is complete by the creases at the side of the nose here; the cartilaginous attachments will sink in. Occasionally you find a deformity that interferes with complete closure of the nostril. In that case the deformed nostril should be simply closed with the finger and the opposite nostril, with one nasal tip, used to secure the vacuum. In using this method for treatment it should only be used in conservative cases. As a rule there is something further of an operative nature that should be done, as Dr. Ballenger suggested. The treatment can be used as an exemplification of the Bier treatment, following any operative measures on the accessory sinuses.

Dr. Holinger:—The aspiration treatment is not the topic to-day, otherwise I might have something to say about it. As a means of treatment it is certainly worth while trying. I would like to ask Dr. Vail if in sawing out the button, it ever happens that the button slips into the antrum and remains there? I think this possibility ought to be excluded, as it certainly would complicate matters. It is not always lack of drainage which is the cause of sinus diseases, especially in affections of the frontal sinus. Very frequently you can enter the sinus with the greatest ease with the largest tube, wash it out completely, and still there are the typical symptoms of headache and discharge. I have operated upon a number of frontal sinuses according to Killian, and certainly I am very critical as to the indications. I often found a large wide entrance and a mucous membrane that was thickened ten to fifteen times its normal thickness with irregular lumps, polyps, etc. I do not see how any one can expect to reduce such swelling by conservative treatment. If recovery were only a question of drainage a tube inserted into the frontal sinus from the nose might be all that is necessary. My experience with the radical operation teaches me that we need not be too timid with this operation in cases where we do not obtain satisfaction from an intranasal operation, because the question is, finally, do you want to keep on treating, and treating for two or three years? Finally, you have to operate anyway. If you have a reasonable amount of patience, say for two or three months, and you do not succeed, there is no reason why you should be afraid to resort to the operation.

Dr. Murphy:—I want to commend the instruments devised by Dr. Andrews for exploring and opening up the sphenoidal sinuses. I am satisfied the more we investigate this obscure sinus—the one that is the most difficult of all to properly investigate and make a diagnosis of—the more frequently we will be surprised by the number of times we will find it diseased where we had not suspected it. I think in the great majority of cases of a chronic postnasal catarrh which do not yield to ordinary treatment, that we have a diseased sphenoidal sinus to deal with. Now the instruments devised by Dr. Andrews are practically harmless. It is next to impossible to penetrate the cribriform plate, which has been a bugbear to us all. We do not always feel certain that we are not penetrating the cribriform plate, and I feel certain those instruments will become quite popular and very practical. In reference to Dr. Beck's transillumination and radiography combined, I think the two should always go together. The one acts as a check upon the other, and where the one proves the other, of course the diagnosis is rendered more certain. In reference to the eye transillumination, I use the larger lamp; Dr. Beck suggests the Hirschman lamp; and in many cases where there is absolutely no diseased antrum on either side, there is a total absence of pupillary reflex. Why that occurs in some cases, and not in others, I am unable to decide, in my own mind, but I feel quite certain we will make a mistake quite frequently if we consider that absence of pupillary reflex is an indication of antrum trouble. In reference to empyemas of the antrum, we do not see nearly all of these cases. We find out that our dental friends see more of these cases than we do, as very frequently they consider it is the tooth that is causing the trouble—being the first symptom—and these patients will ask the dentist to see the case. We are constantly meeting with these cases in which the dentist has attempted to treat them. I have in mind now a case which came in the office, a lady from Louisville, in which the dentist had extracted the tooth and inserted a rubber tube for drainage of the antrum. After a few days the rubber tube disappeared into the antrum and he was unable to extract it. After being there, I think, three months, she came to my office with the history—that the rubber tube had slipped up into the antrum. There was a very foul discharge, and probing through the granulation tissue I was unable to decide whether the rubber tube was there or not. However, opening the sinus through the canine fossa and curetting of the sinus revealed the tube

I feel quite certain that the method devised by Dr. Vail is going to do away with many of the more radical methods, such as opening the canine fossa. I believe in that case had I opened a larger window through the wall of the nasal fossa, that possibly the tube could have been removed and the granulation tissue treated in that way. In reference to Dr. Moore's paper, his conservative method of treating these cases, I think, is very timely. Many of these cases will get well if we give them the proper drainage and wash out the cavity without proceeding to the more heroic method. In nearly all of these cases—in a great many at least—I have had complete success by that method, using the trocar and washing out the cavity.

Dr. Ellett:—I would like to say a word in favor of washing out the antrum through its normal opening instead of by making punctures through the outer wall of the meatus. Of course, that can not be done in every patient, no more than we can probe the other sinuses in every patient, but I believe it can be done in 75 per cent. or 80 per cent. of the cases; at least that has been my experience. It has the advantage of not being an operation at all, and while the washing out may not be as thorough as we give through an opening made lower down, in which case you have two openings —one for the entrance and one for the exit of the solution—you can wash them out very satisfactorily. As far as its value as a matter of treatment is concerned, my experience has not been encouraging except in acute cases, but I believe it will do just as much good as the washing out after puncture and then if we have later to resort to an operation, we can do whatever is expedient.

Dr. Andrews (closing):—Dr. Ballenger remarked that we would be paralyzed at the number of sinuses which the *x*-ray would show opaque or diseased. I admit that I have been surprised at the number of cases in which I have been able to demonstrate the presence of sphenoidal disease. I will not tell you the number of sphenoidal cases I treated during the first ten years of my practice. I will say, however, that during the past two weeks I have discovered four. This is because I have been looking for them recently. I believe I have heard no one mention the air douche for blowing out these cavities. I use this more than I do the washings with solutions. I have felt that air was the natural substance for these cavities, and that a current of air would cleanse them pretty nearly or quite as well as water or a solution, and that it has the advantage at least of not adding moisture, which is necessary for the development of micro-organisms. Regarding the specimens, I would say that part belong to Dr. Fisher and part to the Eye, Ear, Nose and Throat College, and if any of you are interested in them they will be on the table after adjournment, and I will be delighted to attempt to show you how nearly impossible it would be to penetrate the cranial cavity with the knives and probes I have designed.

Dr. Beck (closing):—I look forward to improvements in the method of diagnosis by radiographs which will enable us to show better results. I mean by this we will be able to show the depths of the frontal sinuses by stereoscopic views. Now, in answer to Dr. Murphy, that transillumination and radiography must be combined, I am sorry to say that many times when I get positive findings on a skiagraph, transillumination is negative, particularly so in the frontal sinus, where it is of very little value. The radiograph helps there, although transillumination of the antrum is of service. The reason the pupil does not illuminate in some cases is because of the density of the hard palate. The thinner the hard palate the freer the light goes through the antrum and the infraorbital plate. Dr. Ballenger and I fully agree on the etiology of inflammation of the nose and their effect on the sinuses. I have done Dencher's operation but twice, and I am sorry for it. I prefer the operation of Canfield. The reason why I would not do a Dencher is because of the reaction. It is an operation that is a severe one, and the operation through the nose is better. In regard to Dr. Vail's operation, I have done it three times in all. I have had some tearing with my saw, but Dr. Vail says it is the fault of the saw and not the technic. One point the doctor did not mention. I find I can do better if I operate from the side rather than from the front; then dipping the saw and turning it quickly around helps materially. In regard to Dr. Andrews' paper, that certainly is a timely one, and as Dr. Murphy said, his instruments do away with the bugbear of our work. His instruments are excellent, because with the use of them there is no danger of going into the cranial cavity.

Dr. Vail (closing):—Dr. Beck has just remarked that the trouble with my operation as he performed it was in the saw. He meant the saw which he had. The saw which he used was very faulty in construction; the teeth were long and spicule-like and set obliquely toward him, and it is very difficult to saw bone with it. The saw exhibited here does not present these objections and will saw bone. In regard to Dr. Ballenger's demonstation, we certainly go home feeling repaid for the time we have spent here this afternoon. One point I should like to add to the many valuable ones he brought out is that in all of these cases where pus is coming down from the upper straits of the nose we should make cultures of the pus to see what particular germ is at work—not that it may influence the treatment, but for the scientific interest. The result of the bacteriologic findings might influence the prognosis. If, for instance, you had a case of streptococcus infection, erysipelas might be a complication following operation. Dr. Moore's method—the one which he uses—is certainly the proper one in early cases, and even can be tried in the chronic ones if the patients are timid and afraid and desire to give it a chance. I have nothing but commendation for it. Having failed, however, you might resort to my operation. The first speaker mentioned some objections to the establishment of another opening in the antrum. Practically there is no objection to it. It is plain that if there is pus there it should be let out at the lowest point possible. And he finally concluded by saying, "you must get drainage." There is the key to the situation. That one little sentence is the gist of the whole matter. How are you going to get drainage? The only way I know of is by establishing an opening lower down. Dr. Andrews' method of getting into the sphenoids is ingenious, harmless, and is to be commended. An easy method of getting into the sphenoid I have done repeatedly on the cadaver. Take a large curette, such as is used in mastoid work, pass it through the inferior nasal meatus back to the pharyngeal wall, follow the basilar process toward you with the curette end and slowly lower the handle; keep impinging on the back wall as you raise the distal end of the curette, and you will certainly jump into the cavity. The space between that point where you plunge into the cavity and the cranial cavity is three-fourths of an inch, and in some cases a full inch. So the method is perfectly safe. You can not always see what you are doing in that field, especially when using an anesthetic. In regard to the air douche, as was suggested by one speaker, and also by Dr. McCaw, as being commendable, it certainly is desirable to force out, by means of compressed air, all the liquid left in the antrum cavity, when pursuing the method of treatment for antrum empyema described by Moore. Fluid left there will soon become foul and become a fresh source of trouble in aggravating the disease. A method which is very ingenious was described by which suction can be done with the aid of water pressure. I have used the method of suction by use of compressed air by an apparatus devised by Dr. Dabney. Instead of instructing the patient to say "hic," or some other such word, you can give him a glass of water in which is placed a bent glass tube, and by ordering him to suck the water through the tube and swallow slowly, you can get the desired position of the palate. Any one can be taught that, even a patient who has been spending most of his time in a sanitarium. This method is, however, open to the objection that when he swallows, the Eustachian tubes are opened, thus subjecting the middle ears to suction, which is disagreeable and undesirable. As to Dr. Holinger's inquiry about the button of bone getting lost in the antrum in my operation, there is no danger of that. The saw is behind the button, and after completing the circle, it would not fall into the antrum cavity. It is obliquely cut, and it would be impossible by means of direct pressure to force it in. I have not had the least difficulty in recovering the bone, for it is not so large as would seem from the demonstration here made.

Dr. Moore, Huntington, W. Va., (closing):—I think the principal advantage in the method which I advocate this afternoon is that, in addition to thoroughly cleansing the cavity, we establish drainage through the natural opening. Of course, the opening I make closes up in a very short time, and I do not believe you can get the natural opening thoroughly opened or thoroughly cleanse the cavity by so small a canula as you must use through the natural opening. At least, I have tried it several times with considerable difficulty, and have failed very frequently. I think where I have to remove the nasoantral wall, I will try Dr. Vail's saw. I would like to ask Dr. Vail this: Do you have much secondary hemorrhage with your method? The sawing through the inferior turbinate may cause much hemorrhage. I have frequently had hemorrhage two or three hours after using adrenalin.

Dr. Vail:—I would not call it secondary hemorrhage if it occurred two or three hours after. Secondary hemorrhage would be from three to five days after, which is avoided by packing the cavity.

Dr. Ballenger (closing):—I want to say in reference to Bier's treatment as presented by Dr. Brawley, that I have tried it with a modification of Dabney's instrument for rarefying the air in cavities of the nose, in some cases with success as to diagnosis, and in some cases with gratifying results as a mode of treatment. I recall one case in which there was some muscular asthenopia, and in which the optician who referred the case to me was unable to refract the eyes. I applied the suction after the manner described by Dr. Brawley and with very surprising relief of the headaches and the other symptoms present, after which the case was very easily refracted by the party who sent the case to me. She is again troubled with her eyes, showing the effect of the treatment to be transitory. In discussing my paper Dr. Holinger speaks of the frontal sinus not always being obstructed, and therefore not necessary to remove the obstruction in the vicious circle. I stated there were some twenty-four to thirty predisposing causes to frontal disease, one of which was obstruction to drainage and ventilation. I also wanted to say that the most thorough Killian operation we can devise does not always cure sinuitis. I have done five in the last two years, and in one, which was the most thorough of the five, there was not the slightest relief. The other four cases were absolutely cured and have remained cured to date—not a particle of discharge and fortunately no external disfigurement. In the other case I got disfigurement and no benefit. And I understand that Professor Killian obtains results, both good and bad. The point I want to make is, that no matter what method we use, we do not always get good results. In reference to Dr. Andrews' paper, I wish to say that the front wall of the sphenoidal sinus is not always the point of obstruction of the sphenoid sinus. The middle turbinated body sometimes projects over against the septum at its posterior portion and thus occludes the space in which the sphenoid drains, and, therefore, indirectly obstructs the sinus. Hence in some cases we have to resort to other technic than that recommended by Dr. Andrews. As to Dr. Ellett succeeding in irrigating the maxillary sinus through the osteum maxillare or the accessory openings, that might be true in about 30 to 40 per cent. of the cases. The ostium of the maxillary antrum is very inaccessible. The benefit of irrigation is not always due to the actual irrigation of the antrum. We wash out the infundibulum, the point of obstruction, and thus establish drainage. The statement is made in Logan-Turner's book—one of the best published—and in many other books, that the maxillary sinus is the most often diseased. I do not believe it, as I have been able to so demonstrate. I believe the sphenoid is more often diseased. I believe the ethmoid is more often diseased, and I believe the frontal sinus is more often diseased. Not so diseased that there is need to do an external operation, but the frontal is diseased, as shown by skiagraphy.

THE ESOPHAGOSCOPE AND BRONCHOSCOPE.

SECORD LARGE, M.D.

CLEVELAND, OHIO.

I am not going to read a technical paper, but demonstrate the bronchoscope and esophagoscope on the living.

BRONCHOSCOPY OR TRACHEO-BRONCHOSCOPY.

The first step in the *modus operandi* is the cocainization of the parts, and let me impress on you the necessity of thoroughly cocainizing the larynx and trachea. I think that this is the most important step of the operation. If you do not have your parts thoroughly cocainized, you will have great difficulty in getting the confidence of your patient. I use a 20 per cent. solution and rub it thoroughly into the membrane, using a camel's-hair brush.

I have here two different kinds of bronchoscopes: the Killian and the Jackson. In the Killian, we have here eight tubes of different lengths and different calibers. These tubes are all used with the reflected light.

The Jackson, as you see, has the light in the end, and they also have a tube running the entire length for removal of secretions.

I have often been asked which instrument I preferred, and I must say I like them both; I started with the Killian and naturally have used it oftener, but my advice to a beginner would be to get the Jackson, as the use of the reflected light is more difficult to acquire.

The method I purpose showing you this morning is the direct. With patient sitting in a low chair and head thrown well back, supported by an assistant, the tube is inserted into the larynx, trachea and bronchus, cocainizing as we go along.

The other direct methods are by the aid of the laryngeal mirror, the split spatula or Jackson's slide spatula, or by introducing the finger and using it as a guide.

The indirect method is through a low tracheotomy wound. The different positions of the patient: Either sitting up, as I have shown you, or lying on a table with the head supported by an assistant (as demonstrated by Dr. Jackson) or the patient lying on his side.

We next come to the different kinds of anesthetics: Local, cocain and eucain; general, ether and chloroform. When possible I prefer local anesthesia. In general anesthesia we have been in the habit of giving an initial dose of morphin and atropin hypodermically, one-quarter of the first and one one-hundredth of the latter, one-half hour before giving the anesthetic; we also make an application of a 1 per cent. solution of cocain to the larynx. I have here a very handy apparatus for giving chloroform or ether.

ESOPHAGOSCOPY.

In using the esophagoscope, the pharynx and entrance to the esophagus is cocainized with a 20 per cent. solution.

I have here three different kinds of esophagoscopes: the Mikolitz, the Einhorn, the Jackson. The first one is used with reflected light, and the other two with the light at the end of the tubes.

POSITION OF PATIENT.

I have found by experiments on different patients that the easiest method was to have the patient sit up on a table until the tube, guided by the finger, passes the cricoid, and then have him lie flat on his back, with the head over the end of the table, supported by an assistant. I might say in cases of strictures a bougie is first used to find out its location.

In using the Mikolitz, the Einhorn and the Killian tubes, I use Leiter's panelectroscope as a diagnostic lamp and the Claar reflector, Alexander model, for operating through the tubes. You must also have a rheostat or dry cells with a light controller. It is needless to say that everything must be done under thorough asepsis.

Another little suggestion I would like to give you is to always use a pair of plain glasses, provided you are not already wearing correcting lenses. The patient may cough and some particles might enter the eye. I had a patient cough some mucus into my eye, which contained some of the cocain, and caused a partial paralysis of my accommodating apparatus.

We have used the esophagoscope in fifteen cases. Two foreign bodies, button and safety pin. Eleven in tumors and strictures of esophagus, malignant and non-malignant.

The bronchoscope or trachea-bronchoscope was used in twenty cases. Two foreign bodies, one a peanut shell in the right bronchus, the other a peanut in the left bronchus. Eighteen in cases of compression of the trachea.

As to the accessories, I will have to refer you to the maker's catalogue, as time will not permit me to demonstrate them.

DISCUSSION.

Dr. Keiper, Lafayette, Ind.:—With reference to the retching and gagging, I will give you something which I just got onto a short while back in the applications of medicines to the pharynx or larynx, and that is, namely: If you will have the patients to look down they cannot gag.

Dr. Ballenger:—The point I want to mention grows out of my own experience in a supposed case of a penny in the larynx. I had a skiagraph made of the patient, but on account of peculiar reasons I was unable to see the plate and depended upon the skiagrapher for my information. He telephoned me that the foreign body was at the bifurcation of the trachea. I said to him, "are you sure?" and he said, "yes, I am sure." Upon that statement I attempted to pass a tube through the larynx into the trachea, using the Killian tubes, but was unable, with the assistance of Dr. Stubbs, who also attempted with me to make the passage, to pass the tube into the trachea; we had no tube small enough, the patient being a child 3 years of age. At one time, in passing the tube into the esophagus, I thought I heard a metallic click, but was never able to hear it again, and supposed it was some accidental click that came to my senses, and dismissed it from my mind. I finally gave up the attempt to pass the tube into the trachea, and that evening I went away on my vacation, and a few days later I received the skiagraphic plate, which was shipped to me, and upon examining it I found a shadow on the level with the thyroid cartilages, evidently a penny which was supposed to be the foreign body. It lay crosswise, flatwise, front view—on a level with the thyroid cartilages. In looking at the plate I said to myself if the penny were in the trachea it would in all probability have its edge presented to the front. I wrote to the father that I doubted the presence of the penny in the trachea. And when I arrived home I learned that nine days after I had heard the click, the child had passed the penny via the rectum. Now I want to make the point that we should first be clear as to the diagnosis. First determine whether the foreign body is in the trachea or the esophagus. View the plate personally. Do not depend upon the skiagrapher as I did, as he may mislead you by his statements as to the location of the foreign body. In this case it would have been a sad mistake to have performed lower bronchoscopy, as a glance at the plate showed me the penny was probably in the upper part of the esophagus.

Dr. Murphy:—I thank Dr. Large for the lucid demonstration he has given us to-day. The bronchoscope is going to be an instrument of very great benefit to us in the extraction of foreign bodies from the bronchi or from the esophagus. I had a case in which a young woman had swallowed a small hat pin, two inches in length, with a white head. The radiograph showed the position of the pin very clearly, well off to the right side, evidently off in the bronchus, or even further down. We had no difficulty whatever in passing the tube into the treachea and down to the bifurcation, but we could not pass it into the bronchus. We made a number of attempts to extract the pin with a little hook, as the head of the pin was lying up. However, after working for some time that way we were not able to accomplish anything, and then did a tracheotomy. But that did not help matters and we did not accomplish anything with a tracheotomy further than we could have accomplished with a tube. So we had here subjected the patient to the risk of a tracheotomy, which is in very many of these cases unnecessary. When you can pass the tube down to the bifurcation you can see as much as you can when you do a tracheotomy. Now, the lungs suffered considerable trauma. In passing this hook down and feeling around we accidentally perforated the lung tissue; the small hook got caught in the tissue and I suppose we worked ten minutes trying to extract it. The woman never had any reaction following the whole manipulation. She passed from under our observation, and so far as we know is carrying the pin yet, but it has never caused her any trouble, for she has never returned to the hospital. In another case in which a child had swallowed a jackstone, I was able to extract that under the direct action of the fluoroscope. The foreign body could be seen very clearly, and I simply passed a long pair of curved forceps into the esophagus, while an assistant looked through the fluoroscope. He could see the forceps very clearly, and also the jackstone, which was down well below the clavicle, and thus guide my passing the forceps and grasping the jackstone. I have seen the same thing with a penny. I think where the foreign body is in the esophagus, and of a metal character, little difficulty would be experienced in extracting it with a fluoroscope.

Dr. Iglauer:—I have a case to report that is just the converse of Dr. Ballenger's. The patient, male, aged 37, while blowing on a tin whistle, about the size of a half dollar, bent double, suddenly felt it drop into the throat. He insisted that it had lodged somewhere, he didn't know where, but the doctors in the country did not take much stock in the story. He carried this foreign body for about six months, then he came to Cincinnati. An *x*-ray picture was taken, and Dr. H. J. Whitacre, who had him in charge at that time, from the shadow in the picture, decided that the foreign body was in the esophagus, about on a level with the sixth rib. After consultation with other surgeons, an esophagotomy was performed, but the foreign body was not found. In this case the picture led them to believe it was in the esophagus. The man went home and returned to the city in two weeks. In the meantime the man had coughed up some blood. After consultation with Dr. Whitacre, we decided it must be in the trachea. Owing to the large size of the foreign body, and the fact that I had but little experience with superior bronchoscopy, a tracheotomy was performed. I passed a bronchoscope through the tracheotomy wound and the body was removed with but little difficulty. The patient made an uninterrupted recovery.

Dr. Goldstein:—The case cited by Dr. Ballenger simply impressed on me the importance of the plan of procedure which is adopted in the Killian clinic, namely, before the bronchoscope is used, the laryngoscope is used, then the separable short spatula, and then the deeper and longer tubes. In that way they cover the upper respiratory tract in inspection before the deep bronchoscope is pushed well down for a deeper examination, and perhaps in that way, if it were systematically pursued, we could avoid just such a complication as arose in this particular case. In the matter of *x*-ray work, as far as diagnosis by this means is concerned, it has occurred to me that the fluoroscope should be used at the time the patient is *x*-rayed. A fluoroscopic examination is not always a test of the presence or absence of a body in the respiratory tract or esophagus, and to insure this I think it is absolutely necessary that the entire *x*-ray technic be carried out before a diagnosis is established, because you can find foreign bodies on the printed sheet that you cannot find with the fluoroscope.

Dr. Baldwin, Philadelphia:—I will report one case which shows the tolerance of the throat for foreign bodies. A man carried a full upper plate with three teeth on it three months before it was discovered. He did not report the loss of his teeth, but went to several general practitioners and they did not find the cause of his trouble. Finally one of them passed a tube into the stomach and through this he was fed for several weeks. He finally went to a laryngologist, who found the teeth high in the throat, so located as to be removed with the finger.

Dr. Beck:—Just one point in regard to the technic of *x*-ray. Recently all radiologists have adopted, for the purpose of finding out how deep a foreign body is, the taking of two *x*-ray plates for stereoscopic views. If you will take a picture of a foreign body in the esophagus or trachea, and take two lateral views, and place them in the stereoscope—a large one, of course—you are enabled to tell how far your foreign body is in the eye, or any particular part, from the anterior as well as the posterior side, and therein lies the great point in the diagnosis of foreign bodies in these tracts. Fluoroscopic views only tell you that there is a foreign body; the *x*-ray will localize the level of the foreign body. The difficulty that Dr. Ballenger has, I think, is not a point against radiology, but radiologist, namely, a lack of knowledge in the radiologist. It requires the doctor and radiologist combined to make a diagnosis of the location of foreign bodies.

Dr. Andrews:—I would like to mention a case in this connection in which I assisted Dr. Ferguson of Chicago to operate twelve years ago this fall, before the days of radiography. A patient had swallowed a partial set of artificial teeth. The plate had previously been broken, but he was still trying to use it. I think it had three or four teeth on it. In this particular case there was a large calcareous goiter. He was unable to get the plate from above, and some one had undertaken to push it on down, but without success, and he was compelled to remove the goiter in order to get the foreign body. The patient made a good recovery. Let me emphasize what has been said this morning, not about radiography, but about the radiographer. I have had some experience which has led me to believe that some judgment is required in the taking of the picture, some special training, just as in doing anything else, or in pursuing any other specialty. I have seen some very serious mistakes made simply from lack of knowledge on the part of the man who took the picture. It requires also some experience in the interpretation of the radiograph after it is properly made.

Dr. Stucky:—I would like to emphasize the point made by Drs. Beck and Andrews, and that is, the efficacy of the radiographer in assisting us to understand the radiograph. I am very fortunate in Lexington to have some one to assist me in that way—Dr. John C. Lewis, who made some of the radiographs I showed at Cleveland. Now, I find I cannot tell from the radiograph just how things exist, but with the radiographer to interpret it, the assistance to me is very great, especially in the demonstration of conditions of the sinuses before doing the radical Killian operation.

Dr. Large (closing):—I forgot to show you, when we had the patient cocainized, how easily it is to locate and remove foreign bodies, tumors, etc., from larynx, by the tracheo-bronchoscope. In old people the operation must be done very carefully, as there may be an atheromatous condition of the blood vessels. I am unable to explain the reason why patients do not gag when looking down, but am very glad to hear this from Dr. Keiper. I certainly will put it into practice. Dr. Ballenger spoke of their inability to pass a tube by direct method in the case of a child. I really think that where there is trouble in passing a tube by direct method, it is safer to make a low tracheotomy and pass the tube through this wound. On the one hand you increase your risk by doing a tracheotomy, but on the other you may do more harm by too frequent attempts at passing a tube through the larynx; you may get edema of glottis, so that you are forced to do a tracheotomy or an intubation later on. Dr. Murphy speaks of not being able to get into the bronchi. The right bronchus is easier to enter than the left. If a foreign body is in either bronchus, you must have vent holes in your tubes, or you shut off all the air. The bronchi are movable. Be careful of your asepsis and always examine ends of your tubes to see that there are no sharp points. A number of the doctors have spoken about the value of a good radiographer. In cases of foreign bodies, I always go to the radiographer and examine with the fluoroscope. Dr. Goldstein mentioned Dr. Killian's laryngeal spatula; this is it (shows it), and you may cocainize the larynx and trachea through it. In some cases of foreign bodies, the fluoroscope does not show them at all. I certainly think that this special work belongs to the laryngologist and I think we will be able to hang on to it for a little while at least. My advice to you is to get the instruments, try them first on the dog and the cadaver, and then when you have a case on the human, you will find you will have more confidence.

LARYNGEAL NEOPLASMS IN AMERICA.

J. Leslie Davis, M.D.

PHILADELPHIA.

While the subject of my paper has been announced "Laryngeal Neoplasms in America," it is only so far as they are concerned in the etiology, aggravating complications and confusion in diagnosis that I have touched upon any neoplasms other than carcinoma. "A Brief Inventory of American Opinions on Carcinoma of the Larynx" would be more appropriate.

The magnitude of the subject, the limitation of time for its presentation and the desire for free discussion demand this curtailment of the scope we shall cover.

The history of carcinoma, whether of laryngeal location or elsewhere, is an interesting story in itself. Clouded always in obscurity, regarded from the early centuries to the present time almost universally as a scourge beyond the power of prevention or the chance of cure, it has builded about itself, through distorted imaginations and the insanity of superstition, a barrier through which the bombardment of scientific research has been slow to penetrate.

But passing this and many other points with mere mention, recognizing the thoroughness with which it has already been covered by numerous writers, I shall without apology choose the more practical features that have been equally as well covered, believing that the greatest need of the hour is the iteration and reiteration of principles already propounded, even though nothing new may have been discovered.

I have, furthermore, chosen to summarize deductions from work done within a certain geographical limit, not with any intention of comparison, not with the idea that what concerns the welfare of one country in the working out of its sufferers' "salvation" is in anywise foreign to the interests of any other land where man is found, nor with the delusion that any line of investigation is being or could be carried out here independent of or uninfluenced by deductions, either true or false, which have been brought out in the work of master minds across the seas.

But in this day when index files are so well distributed, and the literature itself so easily accessible, so thoroughly has the work in foreign fields been collected and brought within the range of our reading that any further attempt at compilation would be only to repeat what has already been ably done by others during the past few years. In fact, there are those who become even more familiar with, and more interested perhaps in, what our distant neighbors are doing, less familiar with the work that is being done and more which needs to be done around us, and entirely oblivious to the fact that in the working out of the mighty task each of us may have some part to perform.

Acknowledging anew, then, our debt of gratitude to the cohorts of the conquering hosts wherever they may be, let us proceed to a brief review of our own "Home Guards" in whose rank and file march veterans of the early struggles and younger volunteers alike, who, in spite of discouragement, are waging slow, perhaps, but effective warfare against this dread enemy of all nations.

It is impossible to consider cancer of the larynx and the effort toward its control independently of the study which has been made of cancer involving other structures of the body, since the laws governing its nature and development are the same wherever found, and, while the larynx is one of the less frequent sites, we can point with pride to the work of a few laryngologists in the front ranks of advanced research. To answer, then, "What have been the perils to progress, and what are the factors that facilitate the study of laryngeal carcinoma?" would be, in a large measure, but to answer the same questions applied to carcinoma without restriction as to site.

Roswell Park, in a recent report of the work of the New York State Cancer Laboratory, speaks thus interestingly of the difficulties at the beginning of their undertaking eight years ago, which in reality differs little from the situation twenty years ago: "The motives which prompted its foundation were the inadequacy of the methods applied in the study of the disease, and the entirely partisan views held by practically every investigator of repute. The histologist regarded cancer as a matter of peculiarity of cell structure; the embryologist held it due to peculiarity of cell activity when actuated by hereditary influences or embryonic perversion; the first looking to the cell itself, the latter to its environment and proclivities. The pathologist could advance numerous suggestions, all the outcome of a study of deadroom specimens, through brass tubes, all ingenious, some extraordinarily and incomprehensively so; all displaying familiarity with the minutiæ, and all evincing especially a lack of familiarity with the disease in the living body—a lack of which has characterized most of the published work on the etiology of cancer. Tradition or adhesion to long-established methods had governed all modes of investigation, yet no one had the originality to depart from them. A general spirit of pessimism prevailed in all quarters, the more so as a study of statistics made it appear that cancer mortality was on the increase, at least in those parts of the world which most interest us. This spirit of hopelessness had not exhausted concerted effort, but had rather paralyzed endeavor. It was difficult to secure either men who would devote themselves to such blind work, or money to support them in such efforts."

He further states that finally they succeeded, with the result that there was established in Buffalo the first laboratory ever created for the concerted or deliberate study of the most mysterious disease known to us—one which annually kills seven thousand inhabitants in the State of New York alone. He closes with this personal sentiment: "I can not help feeling that we are much nearer the secret so long sought than we were eight years ago. In fact, I feel that the past eight years have taught us more than the previous eight decades."

The work being carried on by this laboratory in Buffalo is to be strongly commended by every one interested in the welfare of humanity, both professional and layman alike.

In reviewing the literature of American authors during the past twenty years on carcinoma of the larynx—which covers the literature of the world as well—one can not fail to be impressed by the truth of the observation made by Delavan regarding surgical statistics, which could be applied equally as well to data other than surgical, namely: "There can be no statistics possessed of definite value or of conclusive weight until a sufficient number of operators of accredited standing in the surgical world have been willing to faithfully and fully report in all of its detail every case which they have had. All thus far published are not only too promiscuous—representing an aggregation of isolated cases with a tendency to the suppression of unfavorable results—but they are further untrustworthy by reason of confusion of cases, with resulting repetition which tends to invalidate percentages."

Thus in an endeavor to obtain a just estimate of the present attitude of the American profession regarding carcinoma of the larynx, it was my purpose to supplement published records by direct expressions from men engaged in laryngeal work throughout the United States and Canada, desiring particularly personal observations whether in keeping with or differing from the generally adopted conclusions. Unfortunately this course was adopted too late to obtain the good which otherwise might have resulted, since the letters being sent out about August 1 reached their designations after a considerable number of the men had gone on their vacations, and who returned too late in the summer to compile in a complete form the information requested.

However, from a total of about one hundred and fifty letters mailed I had the gratification of receiving over one hundred replies, some of which were from men who have become well known through extensive work and former publications on the subject; others who had reported but a few cases, and still others who, having come in contact with but a still smaller number, had not reported them nor had they preserved records.

Thus, while some of the answers given were from general observation rather than from actual calculations, I feel that they are not without value, particularly points not involving percentages.

For the fuller records given I beg to acknowledge my indebtedness to Drs. J. Solis-Cohen and Joseph S. Gibb of Philadelphia, Chevalier Jackson of Pittsburg, Charles K. Knight, Wendell C. Phillips and Jonathan Wright of New York, Walter E. Murphy of Cincinnati, J. A. Stucky of Lexington, W. L. Ballenger, Joseph C. Beck, Otto Freer and Otto J. Stein of Chicago, Henry L. Swain of New Haven, W. Barclay Stephens of San Francisco, James F. McCaw of Watertown, John F. Barnhill of Indianapolis, and George L. Richards of Fall River.

Since some of the reports failed to mention the number of cases treated, I am unable to give an exact aggregate of the total from which my deductions are drawn—though approximately about two hundred—and it is from these alone that the following estimates are made.

About 90 per cent. of cases were males.

In less than 10 per cent., including distant relatives, could hereditary history be obtained.

The wide range in the observations of different men renders the percentage of heavy smokers and alcoholics difficult to obtain. Cohen states: "I attribute no etiologic significance to either excess," while Jackson, Knight, Murphy and Swain report a majority of their cases addicted to both, particularly smoking.

All agree that the most common of early symptoms observed either by them or by the patients was hoarseness.

Pain was not found to be a frequent symptom before the later stages.

Jackson, Freer and Stephens report cases in which pain was absent throughout the course of the disease.

In the early stages, the vocal cords were reported as the most frequent site of intrinsic involvement; the epiglottis and the arytenoids being the most frequent in extrinsic.

The most frequent causes of death were toxemia, starvation and exhaustion.

None believe that any internal medication, alteratives, etc., will affect the growth of the tumor; nor that any local applications have any permanent beneficial influence. All favor operation if it can be done in the early stage. The most common methods employed are thyrotomy in intrinsic and laryngectomy—partial or complete—in extrinsic. Life is lengthened by operation in a majority of cases, and the patient is made more comfortable while life lasts in a very great majority of cases. The percentage in which life is shortened by operation is very small if done early.

To the question, "From what source is the most hopeful outlook for the future prophylaxis and treatment of this disease?" I received a greater number of "blank returns" than from any question asked; and yet most of those who did reply show a marked unity of opinion, varying only in a slight difference of expression.

Beck: "Through mode of living, prevention of irritation, and early operation when discovered."

Ballinger: "Early diagnosis based on (*a*) constant hoarseness, (*b*) age, 40 or past, (*c*) paralysis of one cord."

Cohen: "Early diagnosis and prompt evulsion."

Freer: "To educate the general practitioner to consider all cases of hoarseness in persons past middle age (especially men) serious. Better statistics, earlier diagnosis and operation."

Gibb: "From bacteriological research, and the discovery of an appropriate antitoxin."

Jackson: "General recognition of the frequently malign nature of chronic hoarseness."

McCaw: "In establishing the etiology and nature of growth."

Phillips, Stein, Richards: "Early diagnosis and operation."

Swain: "The recognition of chronic hoarseness as suspicious."

Jonathan Wright: "From a general study of biological processes of nuclear division."

From these personal communications, then, and from published opinions of the past two decades, we can make the following deductions:

ETIOLOGY.

On this point one might paraphrase an utterance of the prophet of old and well express the situation thus: "Day unto day uttereth speech, but night unto night *faileth* to show knowledge." Various and manifold have been the theories advanced, and yet so frequently have the assertions of yesterday been retracted or refuted to-day that we hardly dare to prophesy for to-morrow.

Animal experimentation, which was for a long time resorted to without any appreciable results in adding to our knowledge of the nature of cancer, has in recent years received a new impulse with the studying of rat or mouse tumors, which seem to be the nearest approach to human cancer yet discovered.

Thus, in support of the parasitic theory, Gaylord cites the instance of an infected cage purchased from a dealer and brought to the New York Cancer Laboratory, out of which sixty or more tumor mice had been taken in the course of three years by the dealer, and in which five cases of cancer developed after the cage was brought to the State Laboratory. He furthermore states that in the State Laboratory, since the beginning of 1905, a typical small spirocheta has been found in all the transplanted tumors thus far examined.

Clowes, of Buffalo, says: "The discovery of the ease with which mouse tumors may be transplanted has led to the development of experimental research in cancer on such a scale as could never have been contemplated five or six years ago."

Bainbridge, in speaking of cancer in general, believes, from the mass of conflicting evidence, the following may be safely adduced, and until the cancer problem shall have been solved will lead no one into danger, namely: that the hereditary and congenital acquirement of cancer are subjects which require much more study before any definite conclusions can be formulated, and in the light of our present knowledge hold no special element of alarm. That the contagiousness or infectiousness of cancer is far from being proved, and that its communication from man to man is so rare, if it really occurs at all, that it can be practically disregarded. And, lastly, that cancer is local in its beginning.

From the standpoint of predisposing causes it is an interesting fact that cancer shows an increase in direct proportion to the advancing scale of civilization; that cancer is least common in the country districts and most common in the larger cities. In other words, the closer to Nature the individual's habits and the less artificial his manner of living, the rarer are his chances for the "contraction" of malignant disease in any form.

It has been well established and generally accepted that the most frequent immediate factor in the production of cancer is some form of irritant, from which stimulation there is brought about a riotous proliferation of cells while free from their natural restraint.

In the case of laryngeal carcinoma, however, it is not only difficult, but oftentimes impossible to attribute its production to any definite exciting agent.

Thus there is no recognized common cause.

That carcinoma of the larynx occurs most frequently past middle life is in keeping with the characteristics of general cancerous conditions, but that a large majority of cases occur in males is one point of difference, and should be borne in mind in cases of suspicion or of doubtful diagnosis.

Some have advanced the opinion that carcinoma of the larynx is brought about through improper or forcible use of the voice, though Jackson observes that in Pittsburg, where chronic laryngitis is exceedingly common, especially among those who use their voices out of doors in all kinds of weather, malignant disease of the larynx is not more frequent than in other large cities with better climate and less frequent occurrence of chronic laryngitis.

Cases are on record which seem to point to tobacco or to alcohol as an unmistakable cause, yet the sum total of evidence does not justify a belief that it plays any important part.

A case now under the writer's observation—age 50 years, who developed an intrinsic carcinoma of the larynx about a year ago—states that for a period of twenty-five years he smoked on an average over twenty cigars a day, and that in recent years he had also been addicted to an excessive use of liquor.

It was this case in which the cause seemed so direct that influenced me to make a special investigation of the part played by tobacco and alcohol, and I confess that it was not without a degree of disappointment that I had to withhold judgment on the ground of insufficient evidence.

Cases are reported in which carcinoma has occurred in connection with or following syphilitic involvement of the larynx, and likewise malignancy has followed the occurrence of benign tumors—a condition, no doubt, which was in either case the result of prolonged constant irritation.

Wright says: "We must allow the possibility of a benign tumor becoming malignant, though the strongest microscopical proof must be required to admit of its probability in any case." To this observation Jackson adds that to demonstrate the benign nature of any growth it is necessary to excise a portion of it all the way down to and including healthy tissue to make sure that there is not a papillomatous or other benign proliferation on the surface of a malignant growth.

The possibility that in the common symptom of chronic hoarseness may be found a clue to a frequent cause is not to be lightly overlooked.

Jacobson observes: "Etiologically, cancer is of intangible dynamic nature, which explains the baffling of investigators; they are looking for something tangible, though the tangible things they find are all secondary."

DIAGNOSIS.

If in the light of present knowledge cancer is to be regarded as local in its origin, and that in this early period of confinement it may be removed by radical operation so perfectly that the chances are overwhelmingly in favor of its non-recurrence, then does the burden of responsibility rest heaviest upon those in position to make the earliest diagnoses.

To me the most promising outlook for the future control of laryngeal cancer comes not from laboratory prophesies of approaching revelations in the nature and cause of the condition, small though our knowledge at present may be; not in the hope of new methods of detection or of signs pathognomonic, though confusion of symptoms may cause deadly delay; not from any fancied faith in the final discovery of a specific, though we would hail such a cure with delight; nor yet from any further perfecting of operative technic, though our present can not be called faultless; but rather in a crusade already begun that shall sweep our whole country in teaching the few principles already proven for making the earliest diagnoses.

To make this most effective is needed the cooperation of every teaching institution, and the author of every text-book that deals not only with the special branch of laryngology, but general diagnosis as well.

Consult the list of our most popular text-books on the practice of medicine, or on general diagnosis, and note how few contain any reference at all to malignant disease of the larynx, and yet it is on these that the majority of general practitioners have depended. From one of the most authoritative of modern books on medical diagnosis I quote: "Malignant disease in various forms may affect the structures of the larynx. A positive diagnosis can be made only with the aid of the microscope. Yet the detection at the seat of the larynx of a growing tumor accompanied by severe cough, by sanious sputum, by signs of destruction of tissue, by perichondritis and exfoliation of the laryngeal cartilages, by hemorrhages and by emaciation, warrants the diagnosis of cancer, whether or not much pain be present. This may be confirmed by the subsequent rapid development of the malignant disease, associated with a musty odor of the breath, distress in swallowing, bloody expectoration, and cachexia."

Such is the clinical picture found in many a case when referred to the "specialist" for treatment.

At least one good lesson is derived from a statement made in not less than ten of the answers received in reply to the aforementioned circular letter sent out to laryngologists: "I am not in position to make any report on carcinoma of the larynx; while a number of cases have come under my observation, the disease was too far advanced when first seen for anything to be done."

Was it the general practitioner's fault that these cases were so far advanced before being diagnosed, if in his college course or textbook reading a method of early detection had not been taught?

Can the patients themselves be blamed for believing that cancer is a disease for which nothing can be done?

Hence we hail with delight the encouragement which has been given to clinical teaching of the "special branches" (otolaryngology, etc.) in some of our leading medical colleges in recent years.

The student of to-day is the physician and the teacher of tomorrow, in the thoroughness of whose education rests the nation's hope for the future dissemination of knowledge.

In this connection we might well call attention to one great peril to progress in the tendency of patients to diagnose their own conditions from "knowledge" gathered from the lay press—which ofttimes is but a wild and erratic creation from the fertile fancy of a dreaming reporter—and then treat themselves with concoctions and cures, which the same press advertises, till all chances for relief through rational means are lost by delay.

No new theory relative to disease, says Bainbridge, can be propounded or research work reported without the probability of exaggerated versions thereof being flaunted in the public press of the civilized world to make upon the minds of the credulous masses impressions for good or ill as the case may be. Reputable physicians, actuated by one kind of motive or another, not infrequently give expression through the medium of the medical press to conclusions drawn from premises that have not been and often can not be verified, and thus confusion worse confounded arises in the public mind concerning matters medical. The public has a right to know the results of all scientific research, particularly as it pertains to health and disease, and more particularly when the public's money defrays the expense thereof. But they should not be overwhelmed with theories formulated in the laboratory, and frightened by specters that oftentimes exist only in the microscope, or rather in the mind of the microscopist.

Chronic hoarseness in the aged is the most trustworthy early warning of possible malignancy that we have.

The characteristic lancinating pain of carcinoma in other parts of the body are not present in all cases of laryngeal carcinoma, and then not before the later stage.

In Stein's admirable paper before the Academy last year we find this statement: "Just because a patient manifests no characteristic cachexia or has no dyspnea or fetid breath, blood-stained expectoration or pain referred to the ears, does not justify one in dismissing the consideration of the possibility of malignancy. To wait for these classic symptoms (these text-book symptoms) is to throw away in many cases the last hope of saving one's patient."

The following observation by the same author is timely: "One of the strongest clinical evidences of malignant involvement I have found to be infiltration. The infiltration is not pathognomonic nor has it any characteristic appearance. Many of these infiltrations have no sharp line of demarcation, but blend imperceptibly with the surrounding structures. At times the infiltration originates deeply or in recesses like a ventricle or is hidden from observation by the cords so that its early recognition becomes impossible; but on account of the pressure it may exert on a branch of the motor nerve or by direct encroachment on the intrinsic muscles there is seen a lazy action of the vocal cords. This sign of lagging during an attempt at phonation when associated with chronic hoarseness should always arouse suspicion and awaken watchfulness."

Syphilitic and tubercular lesions of the larynx may give rise to confusion in diagnosis, particularly the former, in which case the therapeutic test is to be employed. Owing to history, age, location and character of onset and mode of development, tubercular involvement is usually far more easily differentiated.

Simple papillomata are the most frequent benign neoplasms, and likewise the most frequent source of anxiety to the diagnostician. Probatory intralaryngeal removal of pieces of a growth for examination should not be done without consent to radical operation if found to be malignant, and the piece removed must represent all parts of the tumor.

TREATMENT.

This division may be considered under two heads—surgical and non-surgical, either method having advocates equally emphatic in their claims of cure, with cases of non-recurrence to support them in their views, and there are those who condemn all methods alike as futile. "All honor to him who honestly doubts, if through doubt arises investigation, and through investigation conviction," but let us strive not to become prejudiced against any method which has not been fairly tested, nor overzealous for another till it bears the test of time.

The sum total of evidence is to-day undoubtedly in favor of radical operation, thyrotomy being employed in intrinsic carcinoma, and laryngectomy, partial or complete, in extrinsic; and in either case the nearer the operation follows the incipient stage of involvement the greater are the chances of cure.

The question, "Who should operate, the general surgeon or the laryngologist?" has ofttimes been discussed. The argument has been advanced that the one most accustomed to doing major surgical work will employ most careful technic as well as skill in its application. On the other hand, should not the laryngologist who devotes his whole time to the study of a localized field, to its functions as well as its structure, acquire the more perfect knowledge of the part?

It has been suggested that in such operations the laryngologist and general surgeon each has a part to perform; but whoever the operator may be, in the words of Watson, "he must see to it that his enthusiasm is tempered by experience, his courage justified by skill."

The technic of operations does not come within the scope of this paper. Suffice to say that I have been unable to find in the latest American literature anything that could add to the excellency of such methods as those so effectually employed or recommended by Cohen, Crile, Delavan, Halstead, Hartley, Jackson, Keen, Knight, Mackenzie and others.

Of the non-surgical means which have been employed in treatment we shall not attempt to tabulate those already discarded—even though they at some time may have been widely and enthusiastically advocated, but rather give space to a brief consideration of methods which are now being tested.

Treatment by pancreatic enzymes, while discredited by the majority of the profession, must still be regarded as an unsettled question. We shall await with interest the further tests being carried out by one so competent as Bainbridge, who in a preliminary statement remarks:

"Needless it is to say that a few months' trial in a limited number of selected cases can not suffice to forever settle the merits of any method of treatment of cancer."

In view of his name having been used against his will in connection with a reported case of remarkable results from use of trypsin, and widely published by the lay press as well as in medical journals, Dr. Bainbridge says: "In justice to the earnest, truth-seeking element of the profession, both here and abroad, in justice to cancer sufferers the world over, in justice to Dr. Beard and, lastly, to myself, I find it necessary to write as I do now in advance of further definite information. To remain silent longer would tacitly give assent on my part to much that is misleading. The facts as they pertain to the case published do not warrant the slightest enthusiasm as regards cure. While this case is full of disappointment, it presents certain features which give sufficient ground for careful trial of the pancreatic treatment."

T. H. Evans, writing of the presence of sugar in cases of cancer, states that the use of either trypsin or amylopsin will probably be rendered more effective in connection with a diabetic diet. "It is evident that the conflagration of sugar in the body may be destructive to surrounding tissue. We do not wish to supply fuel to the match which we apply in the form of pancreatic ferments." He further observes that the use of diabetic diet should be thought of as a postoperative treatment in cancer, which may delay metastasis and return, and that it is possible that cases of inoperable cancer may be rendered operable by the employment of injections of trypsin or of amylopsin, or of both with an absolute diabetic diet.

The *x*-ray treatment must be regarded of value when applied in connection with or following operative procedures.

Delavan speaks of the *x*-ray as the best means at our command for the relief of some of the worst symptoms of advanced laryngeal carcinoma, such as pain, excessive secretion and fetor.

Of the ultra-violet and the high-frequency rays, and likewise of radium from which interesting results were expected, I have not been able to find any reports of conclusive evidence regarding their value.

The field of therapeutics has been well worked, but without avail, in search for an agent that will exert some influence over malignant growths. Still, there are well-marked indications for therapy which should be met as they arise. If toxemia is the most frequent cause of death, it should be guarded against by promoting free elimination throughout the course of the disease. Exhaustion must be met by stimulation, and starvation obviated through some means of nutrition adapted to each case.

DISCUSSION.

Dr. Ballenger:—I do not feel competent to discuss the paper, but I certainly do want to express my extreme gratification at having had the privilege of listening to it. It means quite a little bit of work on the doctor's part. My own work in laryngeal cancer has been somewhat limited. I have seen a few cases, and operated on a few, by laryngo fissure, total extirpation, etc., and all that. But the important thing is what the doctor emphasizes at the very last, how disseminate knowledge concerning cancer of the larynx first through the profession—though I doubt if that is the quickest way. If we had avenues of approach to the laity—if we could teach them chronic and constant hoarseness is a symptom of this disease—if we could get the laity to know that much, they would soon educate the doctor, and I believe that is the quickest way to solve this problem. I have observed that it takes about fifteen years to get a medical truth disseminated among the doctors, so if we could get the essential facts into the daily papers and magazines, we could quickly inform the laity and through them the doctors. If there were as spirited a campaign on this subject as there is on tuberculosis at present, we could pretty quickly educate the public. I have an opportunity just now of educating a small portion of the public through an encyclopedia of which I am one of the editors, and I have made up my mind I will put the facts in plain language, hoping to arouse some interest. There are ways of using the newspapers legitimately. Organizations should be formed which give you a "trademark," authorizing you to go before the public. That is the quickest way to get around this problem. To wait for the general practitioner and the specialist, and perhaps the general surgeon to be educated, and then the public, means that it will reach the laity perhaps in ten to twenty-five years, and only a few will then know the value of early diagnosis. Let us hope that some organized effort will be made to educate the public as to the early signs of laryngeal cancer, and the value of this knowledge in its successful treatment.

Dr. Murphy:—I am very glad, indeed, to have had the pleasure of listening to such an interesting paper on such a discouraging subject. We all know what feelings of sinking of the heart we have when we have those cases of cancer of the larynx come to us for relief. Unfortunately, as the essayist has remarked, none of the local applications that we are familiar with have given any satisfactory results. The case, like all cases of that character, seems to improve for a short time, but it is of short duration. We have not yet discovered anything efficacious in the way of local application. The only hope, I think, in those cases where the diagnosis is clear—where there has been hoarseness and we can see from the condition of the larynx in the early stages—is the surgical procedure. I believe that is the only one, and as that is quite heroic, it is with difficulty the patient will consent to it until the time for successful operation has passed.

Dr. McCaw:—I think we are to be congratulated upon having Dr. Davis present this matter to us so fully, and he is to be congratulated on the scope of his investigations. He certainly has gone into the subject very thoroughly, although the outlook, as Dr. Murphy says, is very discouraging. Since I communicated with Dr. Davis I have seen one other case of carcinoma of the larynx, of extrinsic variety, which had been seen by some of my confrères in New York; the growth was so extensive that it was advised against doing anything at all. I think, as stated this morning, the outlook for therapeutic measures, dealing with this class of diseases, is in the determination of the etiological factor, and upon that finding depends our success or failure in the future. At present we are inadequately equipped for promising much in the treatment of these cases.

Dr. Bryant:—I would like to ask Dr. Davis to comment on the use of radium.

Dr. Davis (closing):—I would like to say that I am heartily in accord with what Dr. Ballenger says in regard to the dissemination of knowledge, if it is brought about in the right way through the secular press. I believe that oft times much of the blame for the attitude of the press towards physicians rests with the physician. I know how we usually receive a reporter if he comes to secure information about something. Of course, it is our hesitancy in giving out information to reporters. The difficulty of having them report anything that is scientific is that they have not a scientifically trained mind. They are trained to report that which will most attract the attention of the lay readers, and to-day they must put even scientific facts in a fanciful form, and most articles written by reporters on any subject of a scientific nature are more of a fancy born of a fertile imagination than of any element of fact. But, as Dr. Ballenger says, if we ourselves would give out the class of information that should be distributed, then we may better attain the result we seek. I want to say further in regard to the letters I received, there were many who did not report any cases, but I feel that even something was accomplished in sending out the letters, in that several of the men stated their disappointment in not having records of cases that they had treated and that they had observed, and that they had determined to keep better records of their cases. That is one of the chief points of the deficiency in our records—the inadequate information contained in them. In regard to radium, it has been used only very slightly. There are no records of any extensive work that has been done with it. Some report that it has been of benefit following operation, and in some way that the *x*-ray has been supposed to be of benefit following operation, promoting healing and a prevention of recurrence of the growth. I do not recall the name just now of the one who has used both the *x*-ray and the trypsin treatment in combination, but he has not reported any cases of long standing. But in combination of the two he believes that better results will ensue. I think one point we should derive from the answers as to the cause of death—starvation toxemia was the most frequently given. Any method of treatment to be of the most benefit should be supported by every effort to combat the toxemia that exists by a stimulating diet, and prevent exhaustion and preserving every element of strength.

PRIMARY TUBERCULOSIS OF THE UPPER RESPIRATORY AND ALIMENTARY TRACTS,

WITH REPORT OF TWO CASES OF PRIMARY TUBERCULOSIS OF THE UVULA.

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PRIMARY TUBERCULOSIS OF THE UPPER RESPIRATORY AND ALIMENTARY TRACTS.

Tuberculosis, presenting any phase, in any location, is manifestly of such great interest to the medical profession in general that I shall not apologize for this report of two rather rare and interesting cases of presumably primary tuberculosis of the uvula and soft palate, with a brief discussion of primary tuberculosis affecting the upper respiratory and alimentary tracts, mention of several cases not previously reported, and some deductions relative to diagnosis and treatment.

It is vitally important for us all to know and appreciate fully that primary lesions of tuberculosis may and do appear in these unusual places, with, at first, slight, hardly noticeable symptoms, which subsequently may rapidly increase and then indicate pathological changes that may be the cause of much distress and even death.

Granting, if you please, that the diagnosis and treatment of primary and secondary lesions, caused by the bacillus tuberculosis, in the region of the nose, mouth and throat in the late stages are usually in charge of or under the supervision of capable, skilful men, experienced in diseases of these regions, we must still remember that these lesions, often causing insignificant symptoms, are, in the early stages, most frequently under the observation, advice and treatment of the family physician, not especially skilled in this practice, at just the time when the best results from simple treatment are obtainable, and come to the rhinologist, stomatologist and laryngologist when the apparently simple local condition is so far advanced in its progress toward a most severe ulceration, that any treatment will avail little or nothing more than palliation. The cases which I cite below indicate that it behooves us to always give careful attention and consideration to even the most ordinary appearing lesion of the upper respiratory or alimentary tract, that may be evident on just casual examination, remembering that, with no general symptoms, and often with but slight local symptoms, we may have a primary localized tubercular infection, very small in size, with nothing in its appearance to indicate its serious character, that may, if not properly treated, develop and become a large focus of disease, which can not by any means, medical or surgical, be eradicated and may ultimately cause the death of the afflicted individual.

Primary tuberculosis, by which we mean that form of tuberculosis in which the infection is direct, not passing to the part by blood vessel, or lymphatic, and more narrowly, with no other tubercular infection in the same body, as an affection of the upper respiratory and alimentary tracts, is a remarkably rare and unusual condition. when we consider the functions of these parts and the cause of tuberculosis.

Accepting the fact, as demonstrated by Koch, that the cause of tuberculosis is the bacillus tuberculosis, and that this organism is present in man in the most widespread of all diseases, and is the prime factor in disease of animals which are most frequently in contact with man, in as high a per cent. as 30 (Osler), we must feel that the cause is very widely disseminated, and appreciate that it is, indeed, wonderful that the infections by air and food are as infrequent as we are led by statistics to believe.

The special function of the upper respiratory tract is to act as a passage for the respired air, and to prepare the inhaled air to pass into the lungs. In ordinary breathing we inhale about one-half a liter of air at each inspiration (Zarniko), and in ten inspirations, according to Hesse, we inhale in sleeping rooms after cleaning 180 germs, and in the same room with no dust flying only 20 germs, and, according to Petri, in stables 414 germs, and in factories at windows 90 germs. In general, then, this will average about 30 germs per respiration, possibly; about 600 per minte, and 3,600 per hour. Observations by Hesse, Aschenbrandt, Paulsen, Kayser, Bloch, Zaufal, Thompson, Hewlett and others indicate that almost all the organisms that enter the nose in ordinary breathing are arrested by the linings of this organ and the nasopharynx, and that very few reach the larynx except in forced nasal breathing and mouth breathing. From these facts, it seems surprising that we no more frequently have primary infections of the mucous membrane lining the nose and nasopharynx by the bacillus tuberculosis, but it can be easily understood why the parts lower in respiratory tract are not more frequently primarily affected. Dismissing the fallacious belief that we inhale millions of tubercle germs at every inspiration, but accepting the fact that we do receive sufficient numbers of the organisms to cause infections of the nose and nasopharynx, why is it that we so infrequently find primary tuberculosis in these regions? Briefly, the beliefs usually advanced are: The extension of the protecting skin well in upon the septum. where tubercular infiltration or ulceration first shows itself, as a rule; the high degree of nervous reflex present in the nasal mucosa giving rise to violent reaction on stimulation, with secretion of watery mucus or sneezing; the presence of the usual amount of mucus, in which the infection is received, and by which it is washed away, or, if the mucus dries, sneezed or blown away, or passed through the posterior nares and expectorated; the germical action of the mucus, as demonstrated by Wurtz, Lermoyez, D. Braden Kyle, which inhibits the growth and multiplication of organisms; the movement of the cilia of the epithelium toward the entrance of the nose, and, lastly, and probably of least importance, the vibrissæ at the vestibule of the nasal fossæ. Cornet says that, on account of the above-mentioned protections, "the nose affords the worst conceivable conditions for the growth of such bacteria as are not easily endowed with specially active proliferative tendencies, and for the tubercle bacilli particularly." Without question, the presence of abrasions, contusions and wounds on the mucosa of these parts favors the possibility of infection, and, for this reason, the pernicious habit of boring into the nostril with the finger nail or handkerchief, as well as the placing of foreign bodies in the nostrils as is common in childhood, is certainly a cause of abrasions that are an open sesame to the infective agents that may be present at all times, as had been noted by Schmitthuisen, St. Clair Thompson, Kiar, Rosenberg, Felici, and Major.

The upper alimentary tract has for its function the reception of food and drink, with a part of the beginning of digestion of this material, and most unfortunately, in far too many cases, in individuals of bad habits in breathing or those suffering from some nasal obstruction, serves as the upper respiratory tract, as also the receptacle of dirty fingers and foreign bodies in both children and adults. It is needless to remind one that mouth breathing in itself gives the greatest opportunity for infection to occur from the inhaled germ-laden dust, but we must bear in mind that the infection in these cases is almost always pulmonary, primarily. The thick, resistent, squamous epithelium of the mouth, constant movement of the tongue, lips and cheeks, prohibition of growth of tubercle germs by oral mucus (Cornet), passing of saliva and mucus from mouth into pharynx and esophagus, with the infection, before a focus of low resistance may be infected, all afford protection much greater than that of the nose, so that ordinarily no infection can take place in mouth and pharynx, unless the lining membrane is injured, although it has been shown conclusively by Cornet and others that such infection may take place through intact epithelium. Even although areas of irritation and points of trauma are so often present in the mouth and pharynx, making it especially susceptible to infection, when in pulmonary tuberculosis the mouth is constantly bathed in infective sputum, we rarely find secondary tuberculosis of the tongue, lips and cheeks, and the primary form is obviously most infrequent.

Admitting that inspired air with its contained tubercle germs exposes the nose most frequently to infection, and realizing, as stated above, that the trauma quite necessary to nasal primary infection is very often present, and accepting the results of Hesse's experiments which indicate that the organisms that enter the nose are mostly thickly deposited at the entrance of the upper respiratory tract, and diminish in numbers as we pass lower in the tract, so that we find few organisms in the larynx, we can readily see why primary infections of the nose are more frequent than those of the nasopharynx, larynx and trachea, and why the condition manifests itself as an ulceration or infiltration on the septum usually, occasionally on the lower or middle turbinate. Considering the frequency of septal ulcerations, I am inclined to think that it is perfectly fair to believe that there are often primary tubercular foci in the nostril that are considered of little importance, and never diagnosed other than as simple ulceration of the septum, being healed rapidly by curetting and application of proper remedies to the diseased area. This may be the cause for the small number of cases of primary tuberculosis of the nose which we find reported, as often only those ulcerations which are not attended properly, and are soon cured, draw special attention on account of their tendency to progress sluggishly or rapidly, as the condition may be. According to Prota, "tuberculosis of the nasal fossa is a very rare condition, and the primary form without pulmonary complication is still more uncommon. Many of the older writers doubted the possibility of tuberculosis of the nasal mucosa. . . . More recently other authors have admitted that tuberculous ulceration of the nose was possible, but only rarely." In a broad sense, practically all cases of tuberculosis of the nose are primary infections, in that they are direct infections (not by lymph or blood channel), but in the narrow sense, with no other tubercular infection in the body, few of the cases of tuberculosis of the nose may be considered primary infections.

Lennox Brown says: "Tuberculosis of the nose must always be conceded to be both rare and as a primary or early process," feeling no doubt that practically all nasal infections are direct. Cartaz has collected eighteen cases of nasal tuberculosis, seven under his own observation. Of these a great majority had pulmonary tuberculosis. Chiari cited twenty-one observations of "solitary tubercular tumors in the nose," and, according to Shurly, has published four cases of primary tuberculosis of the nose. Coakley says that a few cases of primary tuberculosis of the nose have been reported, and states that the nose is the least frequent seat of tuberculosis of any of the divisions of the respiratory tract. Dionisio, in 1892, found sixty-four cases of tuberculosis of the nose. Farlow has reported two cases of primary nasal tuberculosis. Gradle believes that primary tuberculosis of the nose is occasionally encountered. Hazek collected twenty-seven cases, Heryng ninety cases, and Herzog described twenty cases, his being all primary in origin. Juffinger has written upon nasal tuberculosis, Kafeman has published two cases of primary infection. Knight mentions its rarity and agrees that it may be primary. He feels that the presence of the bacillus tuberculosis in the lesion is the crucial test in diagnosis. Kyle feels that primary infection of the nose presupposes the precedence of abrasion of the mucosa. Mertens, in 1889, described thirty-once cases; Olympitis, in 1890, collected thirty-nine cases; Reidel, two cases, possibly primary; Rethi, one case with no other lesion; Sakolowski and Shaeffer have reported on nasal tuberculosis. E. L. Shurly has never met primary nasal tuberculosis in his practice. Tornwaldt and Willick have made many observations regarding nasal tuberculosis. Without doubt, some of these cases mentioned by different individuals are duplicates of cases appearing in other reports, but I am quite sure that it is safe to consider that certainly one hundred cases of primary tuberculosis in a broad sense have been reported, including those of Sachs, Symonds and Williams, mentioned by Jonathan Wright.

In the nasopharynx, which is not subjected to the trauma that affects the anterior nares so frequently, with the same means of prevention from the infection as the nose, and with a lessened number of organisms on account of the inspired air passing through the nose, but with a better chance of retaining the infection in the crypts of the pharyngeal tonsil than on the relatively smooth mucosa of the nose, it is fairly conservative to estimate that about fifteen cases of primary tuberculosis have been reported, the infection always being in the adenoid tissue in the vault. Dieulafey was the first to point out that there may be tuberculosis of the nasopharynx, as mentioned by Shurly, and Dmochowski, Fischer, Gottstein, Pluder and Suchanneck have all reported cases of primary tuberculosis affecting the nasopharynx. Tuberculosis of the nasopharynx resulting from coughing in persons afflicted with pulmonary tuberculosis is fairly common, I should judge from statements by Baup, Dmochowski, Frankel, E., Koschier, Lartigau, Lewin, Wendt, Wright and Suchanneck, and Nicoll; Lartigau and Nicoll, from their experiments, believe that primary pharyngeal tuberculosis is much more frequent than is usually supposed.

Primary tuberculosis of the lips is so exceedingly rare that it really merits but passing mention. The reasons for its infrequency are obvious. Eiselberg, Platnikow and Scholtz each report a case of primary tuberculosis of the upper lip.

A few cases of primary tuberculosis of the tongue are recorded, but the condition is very infrequent. I personally have seen one case in which the tongue was affected with tubercular infection primarily, *we thought,* from a pipe-stem wound. The condition was cured with difficulty after long treatment. I thought I had found another case of primary tuberculosis of the tongue a few months ago, but the condition proved to be syphiloma. Bonzemet mentions a case of tuberculosis of the tongue, which had disease of the lungs eight months later. Capart speaks of a case. Grasser has reported a case in which a tubercular tumor formed on the tongue at the site of a cigar burn. Laboubene has reported a case with disease of lungs fifteen months later. Mendel has cited a case of lingual ulcer with typical tuberculous lesion in base of ulcer opposite first molar tooth. Schlieferowitsch has added to these two cases of lingual tuberculosis, with no disease apparent in lungs as long as the patients were under observation. Tanturri says that he considers that primary tuberculosis of the tongue is easy to heal. Knight reports a case of deep ulceration of tongue, diagnosed carcinoma, for which the tongue was removed.

In the oropharynx, if the cases I report were not conclusive evidence of the occurrence of primary tuberculosis, we would have to accept the report of Clarke and Fairlee, of the 18-year-old boy, with tubercular ulceration of the roof of the mouth and destruction of the soft palate, with no indication of tuberculosis of the lungs either clinically or postmortem, and consider Isambert's disputed case, in which a young boy had tubercular ulceration of the velum, with no positive signs of pulmonary tuberculosis, although he had scrofula and suspicious rhinitis when he was very young. M. Barbier reports a case of tuberculosis of the velum and tonsils. Bosworth has written that from a practical standpoint primary tuberculosis of the pharynx is not important, although it certainly may occur, as "in any region of the body"; in connection with tuberculosis of the pharynx, his dictum, "it would seem that a deposit of miliary tubercle in the mucous membrane of the respiratory tract assumes a peculiar virulence as the seat of the deposit is in portions near the outer world," assumes considerable interest. Carriere has noted a case of tubercular ulceration in the cheek of a man, not primary in character, although doubtless infected directly. Coakley feels pharyngeal primary infection is exceedingly rare, and thinks infection must occur in abrasion. Chappell has reported three case of tuberculosis of the oropharynx, one of which was primary, in a young woman infected from her sister. The condition ran a rapid course. Inoculation tests were positive, and the tubercle bacilli were found in the sections. Gleason believes pharyngeal infection takes place in an abrasion. Gradle says that the soft palate is more often affected in primary tuberculosis of the pharynx; that the ulceration, when low, causes great pain in swallowing and may cause patient to starve. He feels that diagnosis must be easy, and believes that demonstration of the tubercle bacilli in section makes absolute the diagnosis. In case of doubt in histological examination, he believes that the tuberculin test will determine the condition. Grogler has reported a case of primary tuberculosis, in which the bone part of the palate was affected. Guttmann and Lublinske consider that 1 per cent. of tubercular patients have secondary tuberculosis of the pharynx, and remark on the general tendency for ulceration in this region to be regarded always as syphilis. Heinze has found fourteen cases of secondary tuberculosis of pharynx in 1,226 cases of pulmonary tuberculosis. Kessler has reported a case of primary tuberculosis of the palate and pharynx. Knight says, referring to primary tuberculosis of the pharynx: "Primary cases have been reported, but there is always a suspicion that a deep-seated or limited lesion in the lung may have escaped detection." Kussner has reported a case of palatal infection, not primary. Levy, from a large experience in Colorado, has found that about 1½ per cent. of cases with pulmonary tuberculosis have pharyngeal lesions. In his experience, the course of pharyngeal tuberculosis has been very rapid, three to four months. Morelli mentions a case of primary tuberculosis of the mucous membrane of the gums and cheek. P. Schoetz has reported a case of probable primary tuberculosis of pharynx. Scheh, according to Lennox Brown, has been forced to the belief that the pharynx is affected with primary tuberculosis only apparently, and Brown is in accord with this belief. Shurly says he has met but one case of primary tuberculosis of the walls of the pharynx and back part of cheeks, which were sprinkled with little nutmeg-colored nodules, which by amalgamation broke down into ulcers. Newcomb believes that the uvula and soft palate is most frequent, and that primary tuberculosis occurs in the pharynx in 1 per cent. of all acute tubercular infections. He says that it is not necessary that there be a solution of continuity of tissue for infection, as, according to Strauss and others, infection may take place through intact mucous membrane, and in a recent number of the *Laryngoscope* he calls attention to acute tubercular infection of the pharynx, which on inspection might be easily mistaken for diphtheria. Sleicher has reported a case of primary infection of the pharynx, while E. Sokolowsky has found no case. In 1,307 autopsies Willigk found one case only affected with pharyngeal tuberculosis. Unkerman has reported a case of primary tuberculosis of the velum.

The tonsils seem to be quite frequently affected with tuberculosis primarily. I am rather astonished to learn that Robertson says that "8 per cent. of patients presenting for nose and throat symptoms exhibited primary tuberculosis of the faucial tonsils." The cause for the frequency of infection is the open, deep crypt, which receives and cultivates the infecting organism. Shurly, in his complete treatise, "Diseases of the Nose and Throat," gives some little space in his chapter on tuberculosis of the upper air passages to tuberculosis of the tonsils, and believes that the "reports of Dmochowsky, Hannan, Kafeman, Moritz-Schmidt, Schech, Schlenker and Strassman would indicate that tuberculosis of the tonsils is a common accompaniment of pulmonary phthisis, and that it is very often overlooked." He states, further, that Botey, in his experiments with guinea-pig inoculations, came to the conclusion that there exist certain forms of amygdalitis or hypertrophy of a primary tuberculous character, impossible sometimes to positively distinguish from the common hypertrophy of these organs, with which every one is familiar. Wright says that he is inclined to agree with Hoedenpyl that tuberculous tonsillitis is rare, and that the tonsils are seldom the seat of primary tuberculosis, according to Shurly. Shurly says that he has never met with a case of primary tuberculosis of the tonsil, although his observations cover a long period of time and include the observations of several thousand cases of various forms of tuberculous disease. In a search of the literature I find that Abraham, Hopkins, Koplik, Kruckmann, Orth, Ruge, Sacaze, Schlenker have all reported cases of primary tuberculosis of the tonsil, Koplik mentioning three.

Primary tuberculosis of the mouth and pharynx together has been reported by Gleitsman, who cured the case. Morelli and Stark each mention one case, in which tubercle bacilli were found in carious teeth, with no tubercular lesion in the body.

The inspired air is so well freed from the inhaled organisms by the nose and nasopharynx, the air passes with such rapidity through the larynx, and the protection of the mucous and mucosa is such that primary tuberculosis of the larynx is considered impossible by many, improbable by some, and an actual condition by those who have reported cases, and their followers. It is interesting to note some of the views about primary laryngeal tuberculosis. Lennox Browne, in a chapter on Tuberculous Laryngitis, says: "That evidence of the tubercular diathesis influences a local laryngeal inflammation in a manner eminently characteristic, and at a period long prior to the discovery of equally well-marked symptoms in the lungs, is a fact which the daily observation of those engaged in laryngeal practice establishes as incontrovertible. Whether or not there be tubercle actually developed in the larynx, or what, indeed, is the nature of tubercle wherever developed, the author does not presume, and, indeed, does not care, to decide. Seeing, however, that tuberculosis is a disease primarily manifesting itself more especially in the respiratory organs, seeing that catarrh is one of the most frequent excitants to that disease, and that many catarrhal inflammations of the lungs commence in the larynx, it is at least fair to infer that, in those cases in which the eye reveals what has come to be recognized as tuberculous laryngitis before the ear detects the presence of tubercle in the lungs, the disease has primarily attacked the former organ." This was written in 1877, and twenty years later the author seconded his early opinion by saying: "We may, therefore, for the future, consider tuberculosis of the larynx as a primary disease." In 1881, with Dundas Grant, Lennox Browne prepared two papers to which I have referred elsewhere.

Coakley says: "A few cases of well-authenticated primary tuberculosis of the larynx have been observed, but the majority of supposed primary tubercular infections of the larynx show within a few weeks marked evidences of pulmonary involvement. It is always a matter of doubt whether the condition of the lungs is secondary to that in the larynx or whether, as we believe to be the more common, the evidences of pulmonary tuberculosis were at first so slight as not to be detected on physical examination." Knight, without any question in the matter, says abruptly: "Tuberculosis may attack the larynx primarily or secondarily; in the former case, the process is usually acute; in the latter, chronic. Primary tuberculosis of the larynx is believed by some authorities to be not very uncommon, and is thought to have certain distinguishing characteristics. Twenty-nine cases of primary tuberculosis of the larynx have been reported by Aronsohn, three of which are authentic, in seven the coincident pulmonary lesion was limited and believed to be secondary, while in nineteen the diagnosis of primary laryngeal disease was based solely on clinical signs, which, of course, can not be accepted as conclusive." D. Braden Kyle, in his reference book, "Diseases of the Nose and Throat," discusses primary tuberculosis of the larynx as follows, referring to the lodgment of the bacillus of Koch in the laryngeal structure as the essential factor in this condition: "Whether this be a primary condition arising from infection drawn from without the body, or whether it is always a secondary manifestation from a pre-existing pulmonary consumption, has long been a theme for discussion. With Cohen and others, the author believes, however, that primary infection of the larynx may occur. This view is fully sustained both by theoretical considerations and by postmortem examinations." Newcomb writes: "There is no longer any doubt that tuberculosis may primarily affect the larynx, though many of the cases placed in this category do not present sufficient evidence to exclude the possibility of the existence of some other local deposit to which the laryngeal affection is secondary. So far as the writer knows, no case of laryngeal tuberculosis has ever progressed to a fatal termination without evidence of invasion of other structures, though autopsies have sometimes shown the larynx invaded, but the lungs free." Shurly states, in his work to which I have referred several times: "Tuberculous laryngitis may occur as a primary or secondary affection. Much discussion has taken place upon the question of its *primary* origin, but it must be admitted that it often develops primarily." Wright adds interest to this discussion by saying that "the larynx is by far the most common seat of tuberculosis of the upper air tract, and it is, as a rule, if not always secondary to or associated with pulmonary tuberculosis. . . . This view of laryngeal tuberculosis being always secondary is held by almost all observers, and is proven by the findings of autopsies, there being very few recorded cases of death by laryngeal tuberculosis where pulmonary involvement has not been found. The opponents of this view are very few in number, the most prominent of them being Dr. Gleitsman of New York, whose researches have been extensive, and who reports two cases of primary laryngeal and pharyngeal tuberculosis in his own practice, which were cured. In the report of his cases[1] he quotes Demme, E. Frankel, Pogrebinski, Orth, Coghill, J. S. Cohen, Dehio and Lancereaux in support of his view." He believes it impossible to prove the presence of primary laryngeal tuberculosis without autopsy. He has seen many cases of tubercular laryngitis which he thought were primary, which proved to be combined with pulmonary affection.

I find that de Renzi, Frankel, Josephsohn, Massucci, Orth, Panzer, Trifiletti have reported cases of primary tuberculosis of the larynx, as has also Demme in a child, 4½ years of age, who died of tubercular meningitis, and on autopsy was found an ulceration of the larynx with tubercle bacilli in it. Boylan, Cadier, Dehio, Fischer, Jores, Jullien and Neidert have described apparently primary tuberculosis of the larynx. Capart mentions a case of primary tubercular infection of the tongue with later involvement of the larynx. Tanturri cites one interesting case, in which he made a diagnosis of primary tuberculosis of the larynx, with secondary involvement of the tongue, and cites another case of primary tuberculosis of the tongue, with death resulting from tuberculosis of the larynx.

The only recorded case of primary tuberculosis of the trachea is that of Valette.

No primary tuberculosis of the esophagus has been described. and the secondary form is exceedingly rare, occurring most easily from without.

PRIMARY TUBERCULOSIS OF THE UVULA.

The history of Case 1, which I obtained from the patient, his family physician and a surgeon who operated on him for rectal fistula, is as follows:

Case 1.—The patient, Mr. J. G., of Irish parents, 43 years of age, married, postmaster, and residing in the northern part of Michigan, presented himself for examination of his throat, which, he said, was badly ulcerated. Mr. G. had been married many years, and had a wife and four daughters living, all in excellent health. His wife never had an abortion or still-born child. None of his relatives ever had any diseases bearing on his condition.

Previous to June, 1900, Mr. G. had always been in excellent health, but at that time he was troubled with a rectal fistula, which was operated upon in a hospital in the State of Michigan. The surgeon who operated on Mr. G. for this fistula informed me that, at the time of operation, he found "an ulcerated growth up the rectum which did not heal kindly." This ulcer, he was reminded by the nurse who assisted him, he thought possibly to be malignant, but remembers nothing about there being any throat complication in G.'s case which required attention. While Mr. G. was convalescing at the hospital he noticed "a reddening on one side of his throat, which afterwards became a pimple." A little later the other side became affected similarly. The discomfort occasioned by the irritation of the lesions on his taking acid drinks was sufficient to cause the patient to call the attention of his physician to it, and by treat-

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[1] Journal of Tuberculosis, April, 1901.

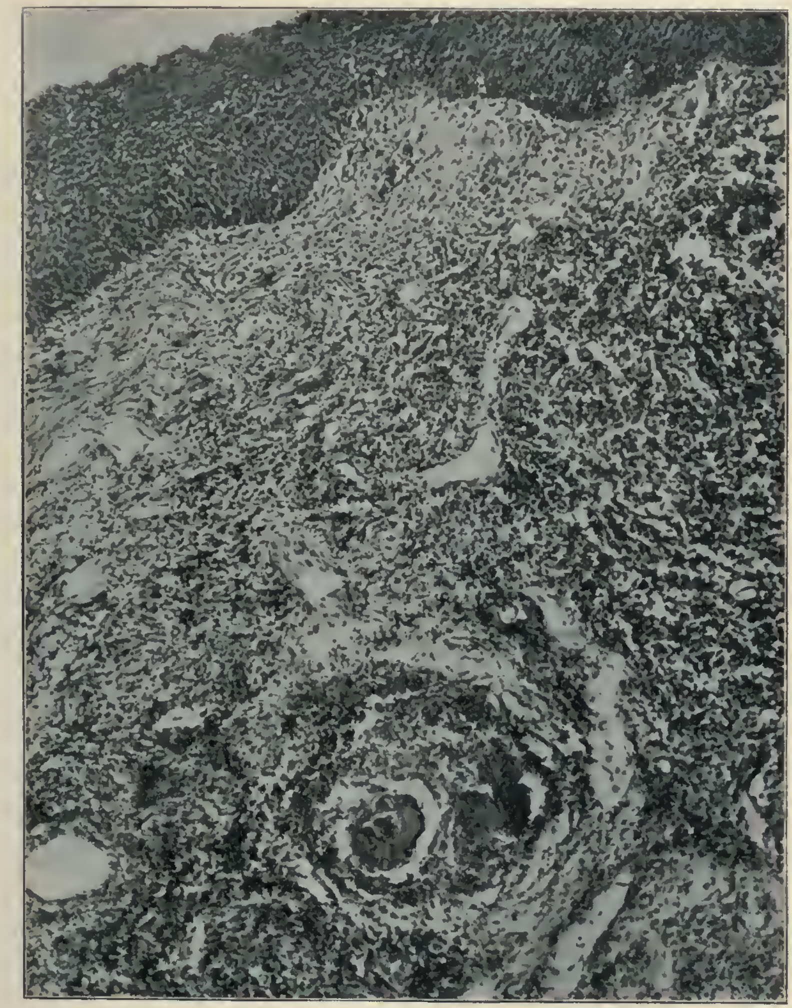

Fig. 1.—Portion of uvula from Baldwin's case. Subepithelial tuberculosis. Epithelioid nodules with caseous centers and containing giant cells.

ment the condition was alleviated and improvement took place. The condition was of such slight interest that no record was made of examination or treatment of Mr. G.'s throat at the hospital. Patient soon left the hospital and attention given the throat was discontinued.

In June, 1902, the patient noticed another sore throat, with a papular lesion on his uvula, similar in appearance to that which he had noticed in his throat previously. This was treated, but not until other papules formed in contiguous tissue, opened and ulcerations resulted, which coalesced and spread over the uvula and on the soft palate. This condition was treated systemically and locally by his family physician, who, regardless of the negative history, suspected syphilis and gave Mr. G. potassium iodid and biniodid of mercury internally, and gave peroxid of hydrogen and borolyptol gargle, with silver nitrate applications locally. It is of interest at this point to note that the patient applied for and received life insurance September 1 of this year, in the examination for which he stated that he had been attended by a physician within the last two months previously on account of ulcerated sore throat. He was recommended by his physician as a good risk, and was granted a policy, which was afterward contested, I believe.

The treatment that G. had been receiving seemed to interrupt the rapid extension of the disease but for a brief period, and then it appeared to be getting so much worse that his physician advised him to go to Ann Arbor and enter the University Hospital.

Patient came to the hospital Nov. 20, 1902. He complained of sore throat that caused constant pain, great annoyance on account of the discharge from the ulcerated focus, and inability to eat anything but liquid food, and that with great discomfort. Further questioning brought out a firm denial of even a possible chance of syphilitic infection, and a statement that every physician who had seen his throat had asked him if he had ever had syphilis; and, knowing that in order to obtain help for his condition, he must disclose all points bearing on his case, he could only assure me that he had never subjected himself to any chance for venereal infection.

The pathological macroscopical appearance of the ulceration of the mouth was noted for me by Dr. Warthin, who described it as follows: The posterior half of the hard palate extending to the alveoli at the sides is indurated and thickly set with small miliary nodules, more or less aggregated into larger papilloma-like masses, and covered with a rather firmly adherent, dirty yellow or grayish pus-like exudate. Anteriorly and at the sides there is a gradual transition to normal mucous membrane, but small miliary nodules are found scattered some distance from the edge of the main lesion. Laterally, the ulceration extends in some places over the alveoli, and a few small nodules are present on the buccal mucous membrane. Posteriorly, the free edge of the soft palate is thickened, ragged and indurated, and presents numerous miliary nodules, a few as large as a split pea. The surface appears ulcerated and is covered with the same grayish exudate. The uvula is long, greatly thickened and indurated, its surface ragged, ulcerated and set with small nodules. A few of the nodules are as large as a pea. Near the base there are many miliary nodules. The pillars of the fauces, the tonsils and the pharyngeal wall are not involved. Dr. Warthin examined the patient and found nowhere on his body any indication of lupus or tuberculosis.

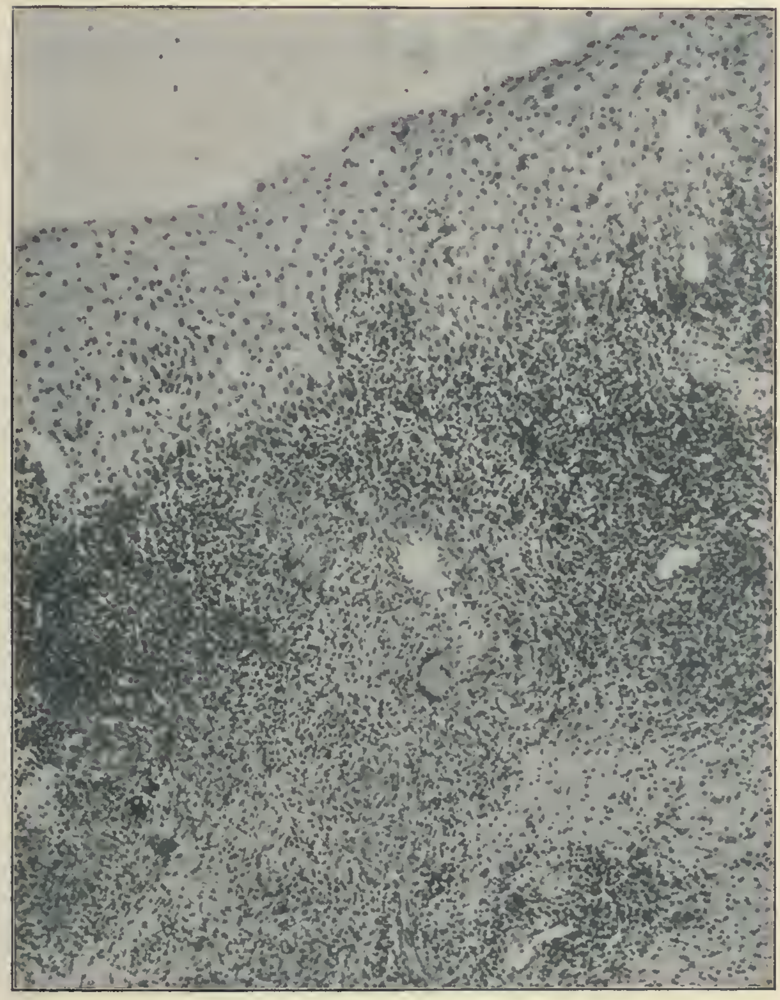

Fig. 2.—Portion of curetting from roof of mouth. Subepithelial tuberculosis (lupus), Case 1.

My examination showed that the nose, pharynx and larynx were free from disease, and that the pillars of the fauces, the tonsils and the tongue were not involved.

The points in the history noted, and the appearance of the diseased part, regardless of the specific treatment administered, and the firm denial by the patient of syphilitic infection, lead to the diagnosis: (1) syphilis; (2) epithelioma; (3) tuberculosis; the preference being in the order named. This diagnosis was concurred in by several clinicians; one suggesting that he would consider simple epithelioma first, and another believed it appeared like lupus-epithelioma, rather than syphilis. Curettings from the diseased area sent to the pathological laboratory for histological examination were reported upon by Dr. Warthin as follows: "Bits of tissue removed from roof of mouth for diagnosis show a characteristic appearance of tuberculosis. May be syphilis, however. I have not had time to stain for bacilli. No evidence, in the part examined, of neoplasm." A second report on the tissue, a few days later, read: "This tissue shows everywhere a subepithelial tuberculosis; a papillomatous growth. Only in one place is there evidence of caseation change." A still later report of the curettings was as follows: "Further examination of specimens from this case shows everywhere a subepithelial tuberculosis, though bacilli have not yet been found. Over the tuberculous granulation tissue is a hyperplastic epithelium, a papillomatous growth; in every way the picture suggests the appearance often found in lupus, namely, a lupus-papilloma. Only in one place is there any suggestion of epitheliomatous change, and this appearance may be explained by transverse and oblique cutting of the papillæ; but, inasmuch as epithelioma often develops in skin lupus, such changes may occur here, although not yet found. The possibility of a so-called lupus-carcinoma must be borne in mind. (A rare and very interesting case.)"

In the meantime I presented the case, one evening, to the members of the University Hospital Clinical Society, suggesting the possibility of the condition being primary tuberculosis, and the interest manifested made it imperative to determine whether the condition was primary in this unusual locality, or secondary to other lesions, especially in the lungs. So at my request Dr. Dock made a thorough physical examination of Mr. G., and gave him a test with tuberculin to determine whether there were any pulmonary lesions of incipient tuberculosis and to see if the patient would react locally or generally to the tuberculin. The physical examination was negative, as was also the examination of the sputum. The tuberculin test was neither absolutely positive nor absolutely negative. To 1 milligram of tuberculin given November 29 we noticed a very slight rise of the temperature to 100.3 on November 30, and I was informed that the patient's throat felt a little better. To 5 milligrams of tuberculin given December 1 we noticed a slight rise in temperature as before to 100.4 and a slight frontal headache. The patient told Dr. Dock that his throat felt a little more sore than usual, but it showed no local reaction. To 10 milligrams of tuberculin given December 5 we noticed a slight headache, with increased soreness in the throat possibly, but no local reaction. The last reaction, if we may call it a reaction, was less apparent than the others, possibly due to an immunity being conferred by the first injections. The tuberculin test, then, may, with reason, be considered doubtful.

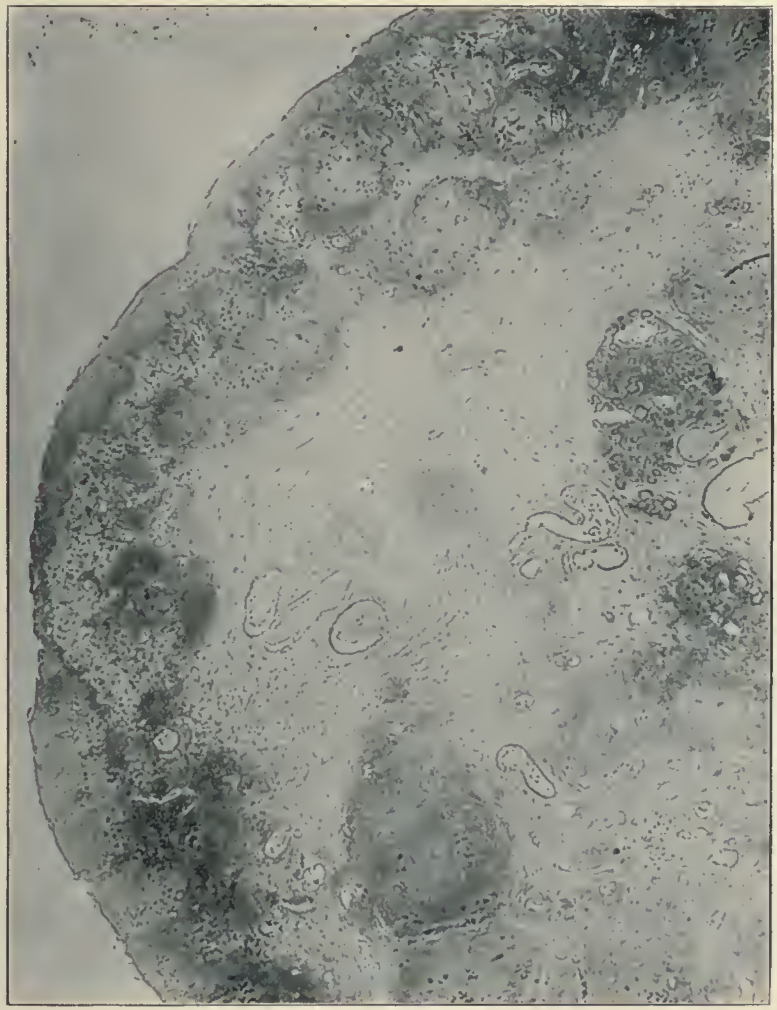

Fig. 3.—Low-power view of part of transverse section of uvula of Case 1. Subepithelial tubercles. Tubercles also scattered throughout deeper tissues of uvula.

Dr. Nancrede examined the patient's right wrist for a swelling present and reported "tenosynovitis, possibly tubercular."

Dr. Carrow concurred with me in my examination and negative findings in the nose, pharynx and larynx.

At this time, as the condition had spread further, getting worse even under drastic treatment, I removed the patient's uvula and sent it to the pathological laboratory and sent material to the bacteriological laboratory for inoculation and culture tests. The inoculation and culture tests were all negative, possibly on account of the small amount of tissue sent to the bacteriologist.

The results of the clinical, histological and bacteriological examinations to this time had not given conclusive evidence that the condition was tuberculosis, although it was evident that, whatever the condition was, it was primary in the location of the uvula. Special stains made by Dr. Warthin for the determination of the presence of the bacillus tuberculosis were reported upon as follows: "Tissue removed from the roof of mouth. Fixed in alcohol, embedded in paraffin, and stained in hematoxylin and eosin. On section there is found the picture of a subepithelial tuberculosis, small nodules of epithelioid tissue without blood vessels, and showing more or less central caseation and containing characteristic giant cells. The connective tissue about the epithelioid nodule is infiltrated with leucocytes and there is a more or less extensive fibroblastic proliferation, causing a lengthening of the papillæ. As the result of such lengthening, there are areas of epithelium apparently extending into the connective tissue. Such appearance may be explained by the cutting, and there are no evidences of malignant proliferation of the epithelium. In some places, the surface epithelium is entirely gone, and the surface of the section consists of a mass of epithelioid tubercles, without ulcerating or caseating areas. Many sections were stained for tubercle bacilli with carbol-fuchsin, but without success. Later a few beaded bacilli were found in the caseated areas. A diagnosis of tuberculosis (lupus) of the mouth may be given, considering the clinical history, apparently a primary condition of the uvula and roof of mouth."

A similar report on the examination of the uvula was as follows: "The uvula was fixed in mercuric chlorid, embedded in paraffin, stained in hematoxylin and eosin, carbolfuchsin and methylene blue. Transverse sections were made of the entire uvula. The epithelium for the greater part is intact, but in a few areas is entirely gone. In some places it is very thin, the superficial layers being absent. Beneath the epithelium and extending entirely around the uvula, there is a zone of epithelioid tubercles, the majority showing caseous centers and containing characteristic giant cells. Some of the nodules are confluent. The tissue about the tubercles is more or less infiltrated with small round cells, and shows a fibroblastic proliferation. The neighboring blood vessels are congested and have thickened walls. Smaller tubercles are scattered throughout the uvula, even in the muscle. The process, however, is pre-eminently a subepithelial tuberculosis. The areas denuded of epithelium present a surface of epithelioid tubercles, in part caseating. In sections a few beaded bacilli are found in the caseous areas."

Besides operative measures to remove the diseased tissue, we used the customary local applications to alleviate Mr. G.'s suffering and to try to prevent the spreading of the ulceration, but to no purpose; in fact, our efforts in his behalf did not seem to improve the condition, and he steadily grew worse, the ulceration spread, and he rapidly lost weight and strength, so he returned home on December 22, as we considered his case to be hopeless.

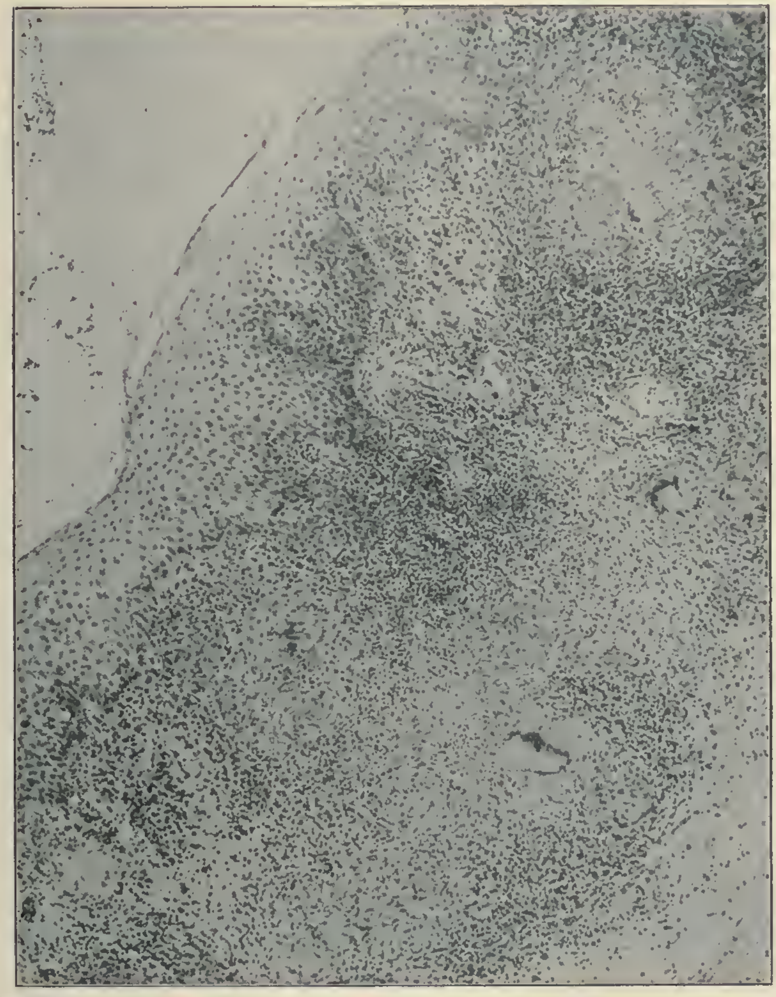

Fig. 4.—Subepithelial tubercles with beginning caseation and containing giant cells. Portion of uvula. Case 1.

On January 28 G.'s physician wrote that since returning home the patient's condition seemed to improve somewhat for a few weeks, which he accounted for by his pleasure at being with his family again; but that this apparent improvement had been but temporary, as he had recently been growing perceptibly weaker and was suffering more pain. He further stated that the ulceration on the roof of the mouth especially had healed quite well, though the surface was broken by pedunculated masses the size of a pea; but posteriorly, and in the region of the tonsils and pillars and upon the alveoli, there was evidence of a spreading of the condition as an

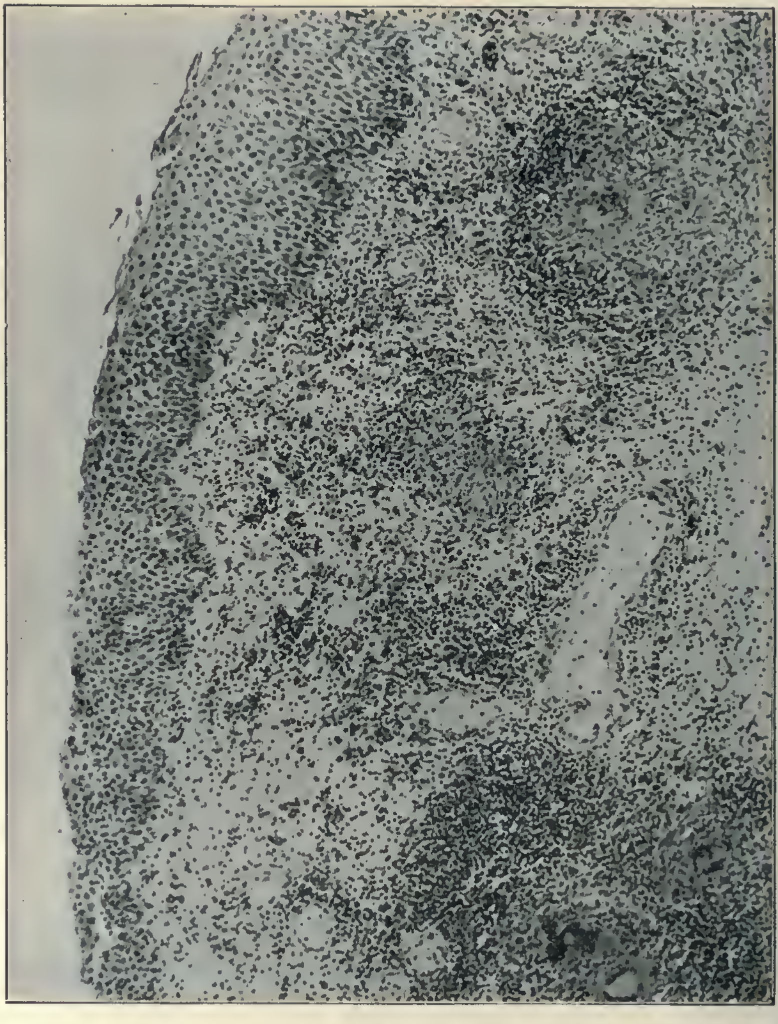

Fig. 5.—Section of nodule from uvula of Case 1. Subepithelial tubercles, infiltration, etc.

open ulcer, covered with a creamy pus exudate. Another communication from G.'s physician, dated March 12, said: "G.'s case is completed. He died February 24 at his home. For about three weeks he suffered from motor paralysis, gradually extending up legs and thighs and body, abdominal muscles and arms, so that the patient was helpless for the last four or five days of his life. Sensation was decreased, so deglutition was easier, and sensibility of mouth, palate and pharynx was diminished. The ulcer seemed to spread along border of gums, down to larynx, trachea and esophagus. His voice was very hoarse, and frequent attacks of coughing brought up enormous quantities of yellow, blood-tinged mucus, almost purulent in character, very difficult to expel toward the last. Gradual failure of strength, impeded respiration, with muscular twitchings, and occasional periods of suffering marked the end. The pulse ran to 150 the last hour, temperature 1 to 2 degrees above normal to subnormal just before death; respiration gradually lessened, with increasing râles, till patient's death. The privilege of an autopsy was denied."

Case 2.—The history of Case 2 I obtained from Dr. D. Braden Kyle of Philadelphia, who, in answer to my inquiry regarding his experience with tuberculosis primary in the region of the uvula and palate, stated that he had seen one case. This case Dr. Kyle courteously allows me to report with the history of the case I have just detailed. In two communications received from Dr. Kyle he says: "I have seen a number of cases in which the pharynx and tonsillar folds were involved in tuberculosis secondary to the pulmonary lesion, but I have only seen one case of primary tuberculosis of the uvula and soft palate. The ulceration, which was proven to be tuberculous by microscopic examination of the secretion and scrapings from the part, began on the soft palate and uvula, as near as I could obtain from the history, involving both structures when first noticed. There were no pulmonary symptoms until eighteen months later, when a slight lesion was discovered in the right apex. In the meantime the tuberculous lesion had involved the pharynx and later the epiglottis and larynx. The patient was at once sent to Colorado and died six months later. I think that primary lesions of the uvula and soft palate are, indeed, rare, although I think primary lesions of the larynx are more common than is generally supposed." In answer to my request for further data, slides and any additional information regarding the case, Dr. Kyle wrote: "I did not make any inoculation tests, either in animals or test-tubes. The microscopic examination of scrapings showed so clearly and distinctly the tubercle bacilli that I did not think it necessary to make further investigations. Unfortunately I did not retain the slides, but the microscope fields were filled with tubercle bacilli, and by contrast stain showed many other bacteria present. Personally, I have not the slightest doubt as to the diagnosis, although it was not confirmed by the tuberculin test or serum cultures."

In a careful examination of the literature on the subject of primary tuberculosis of the uvula and palate, I find one undoubted case, reported by Baldwin as "tuberculosis of the uvula" in his article, "Some Unusual Localizations of Tuberculosis." Baldwin's exhaustive examination of the literature up to the time of this report failed to reveal any case reported in which the uvula had shown tuberculosis. To quote from his paper: "Although tuberculosis of the pharynx and larynx is of common occurrence, I have been unable to find any case reported where the uvula has shown any tuberculosis. This case, then, is unique in that it is one of the first, if not the first, case of tuberculosis of the uvula." Baldwin gave me personally a brief description of this case and the findings as follows: "A woman, aged between 50 and 60 years; good family and personal history, with no evidences of tuberculosis, came to Dr. Carrow for a small nodule on the uvula, which was removed and proved to be chronic granulation tissue (tubercular). The uvula was then removed *in toto* and examined. Microscopically, just beneath the mucous membrane was a granulation tissue very poor in blood vessels. Deeper in the parenchyma of the uvula were small masses of epithelioid tissue containing no blood vessels. Scattered through these masses and making up a large part of some of them, giant cells, with peripherally arranged nuclei, were found. Sections of the uvula stained with carbol-fuchsin showed a few tubercle bacilli in the giant cells and in the other epithelioid tissue."

I wish to lay especial stress upon the fact that this patient, whose history Dr. Baldwin gave me, at the last report was alive and well, with no indication of having ever suffered with tuberculosis of the uvula.

Dabney reports having seen "one of the rare cases of tuberculosis beginning in the upper part of the throat." He says that he has seen two cases and possibly one or two others. In his reported case, he found the lesion confined to the roof of the mouth, a typical ulceration causing considerable pain and interfering with the patient's nutrition. Examinations of the scrapings from the ulceration were negative, the diagnosis being based then on the macroscopical appearance. Examination of the patient's chest by several physicians failed to reveal pathological condition of the lungs, till ten days before death, when a deposit in the apex of one lung was discovered. In answer to a letter from me, Dr. Dabney wrote me that he had seen another case, unquestionably tuberculosis, apparently primary, with this history: "The patient was a youth, about 20, whose mother had died of tuberculosis when he was an infant. Her trouble is said to have begun in the throat, probably laryngeal. In the young man's case, before any symptoms could be detected in the lungs, there appeared a worm-eaten ulceration on the soft palate, gradually extending over its surface and down upon the pharyngeal wall. It was attended with great pain in swallowing, steady loss of weight, and a temperature ranging from normal to 103 or more. The appearance of the patient was typically tubercular, and microscopic examination confirmed the diagnosis. He was sent to Asheville, N. C., but steadily lost ground and died within a few months after the first evidence of the disease appeared."

Lennox Browne has described a case of secondary tuberculosis of the uvula with an accompanying illustration, which has been largely copied in other text-books.

Grunwald, in a personal communication, referred me to his "Atlas and Epitome of Diseases of the Mouth, Pharynx and Nose," in which there is a good illustration of secondary tubercular lesion of mouth and hard palate, which from the history of the case is doubtless a secondary infection to a possibly primary nasal lesion, "the infection probably traveling from the floor of the nose by way of the lymphatic clefts of the incisive foramina."

Ingals several years ago wrote that he never had seen either primary or secondary tubercular infections of the uvula, nor primary of the palate, but had seen secondary infections of the palate and fauces.

Knight wrote me that he had observed several cases of tuberculosis involving the uvula and palate by extension from the pharynx, but no case in which these regions were primarily the seat of tubercular infection.

D. Braden Kyle, in his excellent work, "Diseases of the Nose and Throat," says that ulceration of the uvula occurs as a primary infection in tuberculosis. The tubercular involvement may appear as small, wart-like excrescences which go on to ulcerations, so arranged that a peculiar club-like formation is given the uvula.

Moussous has reported a case of primary tuberculosis of the uvula, tonsils and anterior pillars of the fauces.

Phillips, after mentioning that primary disease of the uvula is rarely seen, states positively that lupus does not primarily affect the uvula, but papillomas are occasionally seen, being usually dependent upon syphilis or tuberculosis.

Prota reports a case of primary tuberculosis of the nasal mucous membrane, with secondary involvement of the uvula and soft palate and pharynx. He says in part: "The uvula was in part destroyed. The soft palate was infiltrated with small miliary nodules which were roughened, rose colored and slightly translucent." I note that Prota's bacteriological tests were negative, but that he obtained a positive tuberculin reaction. Although the microscopical examination here was proof of the condition being tuberculosis, he points out the difficulty of diagnosing tuberculosis from syphilis, even microscopically, mentioning the fact that in only eleven of twenty-seven cases of tuberculosis of the nose were tubercle bacilli found.

Sir Felix Semon some time ago wrote me that he had never observed a case of undoubted primary tuberculosis of the uvula and soft palate, but without referring to his case books he would say that he had seen about twenty cases of secondary infections of these parts in thirty years' experience. I was unable to get a copy of his communication to the Laryngological Society of London on "A Case of Obscure Lardaceous-looking Infiltration of the Uvula," etc.

Shurly wrote me that he had seen one case of primary tuberculosis of the uvula, which spread thence to the lymphatic system of the lungs. He had in mind no case in which the condition was confined to the palate alone, but had seen several cases where this region was involved.

Swain, in a long experience, a few years ago, had not seen any cases presenting tuberculosis, either primary or secondary, of the uvula or soft palate.

Treitel has reported a case of primary tuberculosis of the palate.

Wright has reported a case of primary tuberculosis of the uvula and pharynx.

From the fact that Birkett has mentioned in his report of the case of primary lupus of the pharynx that "the pharyngeal aspect of the uvula shows two distinct nodular infiltrations, each about the size of a lentil, the mucous membrane covering these being quite intact," I have reserved the mentioning of this case until I described affections of the uvula, not wishing to discuss tuberculosis and lupus. Birkett says: "What is necessary in all cases of this nature is to establish the circle of evidence, namely, presence of the tubercle bacilli; definite results from inoculation; and, I think one may say, the reaction from the use of tuberculin." The bacilli were found; inoculations were positive, as was also the tuberculin test.

CONCLUSIONS.

Primary tuberculosis of the upper respiratory and alimentary tracts is not such a common condition that we can generalize or draw many conclusions from the relatively small number of cases reported, and the few that we may meet in our own personal experience. We can not help but note that many of the presumably primary infections are at some time later disproved by indications of previous or concomitant general infections, or marked local infections which must have been overlooked at the time of earlier examination, on account of its presence being undemonstrable. The concensus of opinion is that these infections appearing in the upper respiratory and alimentary tracts, whether primary or secondary, are very severe, and run their course in a short space of time, but that if seen and treated early in their course the chances for cure are good.

These lesions are not especially easy to diagnose, I am constrained to say, from my experience with the case whose history I have given, as the clinical picture of tuberculosis is much like that of syphilis or neoplasm. This clinical picture not being characteristic enough to prove the condition either tuberculosis, syphilis or neoplasm, we must rely upon the microscope to differentiate by histological structure of the tissue, presence or absence of organisms in the examined tissue, or demonstration of the organism in culture medium or animal inoculation tests. Personally, the fact that the bacillus tuberculosis is found in the removed tissue is proof positive to me of the nature of the condition. The only practical clinical test, to my mind, is the thorough pushing of antisyphilitic treatment, although I am well aware that many feel that the tuberculin test is infallible in its demonstration of the presence or absence of tuberculosis, but often the course of such infections is so brief that the test by treatment would be unwarranted, to say the least.

Could our old friend, Aretæus, the Cappadocian, have had in mind primary tuberculosis of the upper respiratory and alimentary tracts when he wrote, in 50 A. D., "The postponement of medical treatment is a bad thing, for by procrastination they pass into incurable affections, being of such a nature that they do not readily go off, if they once attack, and if protracted by time they will become strong and end only in death"?

When we consider the prophylaxis of primary tubercular infections of these regions, we must have in mind the factor in prophylaxis of tuberculosis in general. As at the present time tuberculosis is only theoretically preventable, and for obvious reasons will not be practically preventable for some time, it is necessary for us to consider the prophylaxis largely from the point of the condition of the locality affected and those things that are liable to increase the chances for such infection. Dismissing as not feasible, then, the general prophylaxis of tuberculosis, we must expect the presence of the bacillus tuberculosis in the inspired air of practically every individual at some time during the day, and plan, if possible, to prevent the organisms, which must be present practically at all times in the nasal and oral mucus, from infecting the membrane itself, by proper attention to nasal and oral hygiene, and by the correction of any local or general conditions which may determine these particular regions as an easy avenue of infection. I am fully confident that we often err when, in our converse with tubercular patients, we do not continually keep before them the fact that they are a menace to all those with whom they come in contact, or even those who may never see them or know them, providing they do not attend properly to the prevention of the spreading of their infection.

Children and adults should be instructed that danger lies in careless picking at the nose to remove the little crusts that may accumulate in the nares, and that the nose should receive as careful attention in its daily treatment and care as any other part of the body. Mouth breathing should be corrected. Foreign bodies should not be put into the nose and mouth. Promiscuous osculation should be abandoned. The use of public eating and drinking utensils should be limited. Whenever slight chronic conditions of hyperemia of the mucous membrane exist, they should receive the attention of the physician, who only should advise the douche or spray or gargle that may be needed. If all this were carried out, the result would be that systematic and complete and careful examinations of the upper respiratory and alimentary tracts would be made at regular intervals, and any suspicious conditions could be investigated at an early date.

Although none of us will hardly agree with our preceptor, Galen, who, in 140 A. D., said: "Ulcer (ἕλχος) of the lungs was less frequent than ulceration of the larynx and trachea," we must believe that ulcerations and other affections of the upper respiratory and alimentary tracts, caused by the bacillus tuberculosis, are sufficiently frequent even as primary conditions to merit the careful consideration and attention of all the men who are working toward the common end of alleviation of pain and suffering, and, better still, the prevention of those conditions that cause pain and suffering and even death.

I wish to acknowledge with thanks the assistance rendered me in the preparation of this paper, by direct help in the attention given the patient when he was in my care, or by laboratory work with the examination of material from the patient, or clerical assistance in investigation of the literature on the subject of primary tuberculosis, of the following of my colleagues. In preparation of the paper I have consulted freely the text-books mentioned in the table of references, Nothnagel's Encyclopedia of Practical Medicine, Index Medicus, Index Medicus Novus, and various journals of medical science: Drs. Frederick Baldwin, Fleming Carrow, George Dock, Charles B. Nancrede, Alfred Scott Warthin, A. W. Wilkinson, D. Braden Kyle, and those from whom I have received personal communications.

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DISCUSSION.

Dr. Goldstein:—I think it is pretty generally conceded to-day that primary tuberculosis of different areas of the upper respiratory tract may occur, and Dr. Willis' paper is simply another contribution to the selection of cases that are gradually being heaped up in literature. I recall a case reported before this body eight years ago of primary tuberculosis of the larynx at a time this question had been thrashed out, and there was a most unmerciful criticism of such a possibility. At the next meeting of this body I shall have a postmortem larynx to exhibit of a case of this kind. Four years ago I reported four cases of primary tuberculosis of the ear, and since that time the literature is becoming richer in the whole field of primary tuberculosis. I think we are pretty well agreed to-day that such a definite form of tuberculosis, located primarily in the upper respiratory areas, may exist.

Dr. Iglauer:—I should like to ask Dr. Willis if it is not possible that the fistula in the rectum may have been of a tubercular nature, and if the infection of the pharynx could not have been by metastasis. Further, how does he explain the absence of much reaction even with large doses of tuberculin?

Dr. Willis (closing):—Dr. Goldstein says that he thinks that we are all agreed that primary tuberculosis of the upper respiratory and alimentary tracts does exist. If he will read the remainder of my paper, which time did not permit me to finish, he will find that such is not the case. There are two definitions of primary tuberculosis: One, in the narrow sense, in which there is no other tubercular focus in the body; the other, in the wide sense, in which there may be another focus in the body, which is not, however, the origin of the second focus by metastatic extension by blood or lymph channel, but is the seat of infection in that the material from this focus is expelled and directly infects the second focus. Dr. Iglauer suggests in regard to the fistula in the anus and ulcer in the rectum, that they may have been due to infection by tubercle organisms, and that then these points may have been the original seat of the primary infection. He will note in my discussion of the case that examinations by that skilled diagnostician and clinician, Dr. George Dock, resulted negatively, in so far as the finding of any bowel condition was concerned. The result of the operation for the fistula was satisfactory. Just by chance, I learned of that condition in the rectum, as the patient did not mention it if I remember rightly. The surgeon who operated upon the fistula in ano wrote me that he believed that he remembered that one of his nurses in the hospital said that she thought that he said that there was an ulcer "up the patient's rectum which did not heal kindly." Provided there was such an ulceration, and granting that the ulceration was tuberculous, I do not for one moment believe that infection could have taken place from this point to the uvula, by lymphatic or blood current, as certainly foci of lower resistance than the uvula must have been traversel by blood or lymph stream, as is anatomically possible. Referring to the tuberculin test, I must say that I certainly do not consider it an infallible test for tuberculosis. Unless the result, which was more or less apparently due to the injection of tuberculin, which I mentioned in the discussion of the case, constitutes a true tuberculin reaction, I am positive that the reaction to tuberculin injection as an absolutely sure test for tuberculosis is fallible, for you will note that as the organism causing tuberculosis is present in typical tubercular tissue histologically, the condition is proved beyond doubt to be tuberculosis. I wish to thank the gentleman for their discussion of this paper.

MALIGNANT GROWTHS OF THE NASOPHARYNX.

A REPORT OF TWO CASES.

Kate Wylie Baldwin, M.D.

PHILADELPHIA, PA.

It is not my intention to go over the subject as a whole, but it is desirable that every case be put on record, and I wish simply to add two to the list.

As with malignant growths in other parts of the body, the early symptoms are not usually sufficient to make the patient ask advice, and the condition is not discovered in time for surgical interference entirely to eradicate infected tissue.

Malignant growths are not, in themselves, painful. It is only when they encroach upon sensitive organs that the patient receives warning, or when located where friction or other irritation causes hemorrhage. Fortunate is the patient who has an early hemorrhage, as he is apt to seek relief in time for operative interference to be of service. In the early stages sufficient pain to demand operation is seldom experienced.

The first symptom of a growth in the nasopharynx that attracts the notice of the patient to an extent to cause him to seek relief is usually earache. The headache, if any, is much later. A fulness in the throat or an obstruction to nasal respiration is often not observed by a busy man or woman. It is well for the patient if his first hemorrhage is through the nose; otherwise he consults the general practitioner or the chest specialist, as he is usually positive that the blood is from the lungs or stomach. In either case much valuable time is lost. Any specialist will first eliminate the region of his own specialty before sending a patient to another.

Small quantities of fresh blood from the mouth should always suggest an examination of the teeth and gums. I am frequently surprised at the almost total neglect even to think of them as a cause. Any digestive trouble, a general septic condition or almost any trouble in nose, throat or ear demands a careful inspection of the gums and of every tooth. Two years ago I knew of a young man who lost his life through hemorrhage from the gum. Four months ago a lady came to me who, on the street, had suffered what she described as a severe hemorrhage from the lungs, even locating the exact portion of the left lung from which it came. Examination proved to me that the blood came from the nasopharynx, where there was some thickening and marked dilatation of the blood vessels, with the surface at the time covered with blood, while the lower part of the throat and larynx were entirely free and the chest gave no evidence of being involved. This patient has, at different times, had both breasts removed without return of the growth in either side. I do not say there is a malignant condition in the nasopharynx, but I shall keep her under careful observation.

Case 1.—The first case I report here is that of a 16-year-old Russian boy. I first saw him in September, 1905. In June he had been in a hospital one week with nasal hemorrhage. Bleeding recurred September 25, and there were three distinct hemorrhages in twenty-four hours. When not bleeding, congestion rendered nasal breathing impossible or difficult. At no time had there been pain in the ear, sore throat or headache. Had it not been for the hemorrhage, doubtless he would have gone several months longer before having an examination. I think there had been no examination of the nasopharynx in June.

Examination.—No antrum or frontal tenderness; right ear normal; left ear, considerable retraction of the tympanic membrane; no marked loss of hearing. Odor of ozena; nasal fossæ free anteriorly; septum deflected to the left with ridge at base on both sides; blood on the floor of the nose; teeth good; palatal arch high, moderately narrow; velum dry and mucous membrane inflamed; tongue coated; uvula relaxed; faucial tonsils slightly full; lingual tonsil and larynx normal; pharyngeal wall covered and choanæ filled with clotted blood. After cleaning, I found a growth springing from the neighborhood of the left inferior turbinal, reaching to the upper edge of the left choana and nearly covering the opening of the left Eustachian tube; left choana entirely closed and the right nearly so. After contracting the right inferior turbinal, the growth was distinctly seen through the right side of the nose. Examination of the blood showed practically normal coagulation in four and a half minutes. The color was good and it flowed freely.

To improve the general condition, that he might the better stand the removal of the growth, he was put on iron with plenty of good food and fresh air. Suprarenal powder was used through the nose. He had repeated hemorrhages and the general condition suffered accordingly. The growth increased very slowly.

December 7, under local anesthesia, I removed the entire growth. I was able to constrict the base with a snare. The tissue was too dense for the wire to cut, but it did prevent hemorrhage. With a small, strong spoke shave I divided the pedicle back of the snare. This was followed by a terrific hemorrhage. I quickly delivered the growth and carried into the nasopharynx a firm plug of sterile wool yarn, meeting it from the front with a strip of ham fat with which I filled the left side of the nose. Everything was at hand and by disregarding the flow of blood this packing was quickly placed and the hemorrhage controlled.

The patient was put to bed with the head high. The bowels were thoroughly moved with calomel. He was given liquids freely, but no food that required chewing. On the second day I loosened the postnasal packing, leaving it so that it could be drawn quickly into place should there be bleeding. As there was none, twenty-four hours later I removed the postnasal packing and a little of the fat, leaving the remainder to care for itself, which it does without any danger to the patient.

Four days after the operation he was allowed to go home, with instruction to keep perfectly quiet and, should there be any bleeding, to send for me, but also to call in the nearest physician. I gave him a note to give to any physician called in, telling what the operation had been and suggesting a postnasal packing.

For over four months there was no return of the hemorrhage. The growth proved to spring from the lateral wall of the left choana. The surface was smooth and there was all appearance of a thorough removal of the pathological tissue, which proved to be an endothelium, probably sarcoma. It healed promptly and for three months there was no indication of return. Then there appeared a general thickening which extended beyond the original site. Six weeks later there was some bleeding which was repeated several times, but no operation was indicated which was not too radical to be safe.

The boy disappeared and I took it for granted that he had had a fatal hemorrhage. I left the city in July and heard nothing from him when I returned in September.

Last May, nearly two years after the first hemorrhage, I looked up the family that I might finish the history of the case. Much to my surprise, I found he was not dead. During my absence the hemorrhage became so severe and frequent that he went to the hospital, where it was found necessary to operate. The boy did not know who operated, but said the growth was larger than the first one and softer and followed by a much greater loss of blood and much greater prostration.

He was kept in the hospital two days and went home in a very weak condition, having had, from his point of view, nothing to eat. Two days later he suffered the most severe hemorrhage he had ever had and very nearly lost his life. He was taken to the nearest hospital in a patrol wagon and remained a week, returning home to be in bed for nearly three months. This was seven or eight months after the first operation.

Examination of the throat May 27, 1907, showed the local condition very good, with only slight roughness and a small nodule at the original site of trouble. The thickening extended to, but not beyond, the median line. The boy was thin and not of good color, but claimed to feel fairly well, though not strong. He was unable to do any heavy work and had done nothing until three months previous to the time I saw him.

The *x*-ray was suggested both before and after the first operation, but it was decided that there was little benefit to be expected and much danger of its causing severe hemorrhage.

CASE 2.—A woman, 42 years old. The father, one sister and an aunt died of consumption. The cause of the mother's death is not known. The personal history is unusually good. She had none of the diseases of childhood; vision in right eye never so good as in the left; for the last ten years has had attacks of hay fever.

In the summer of 1904 she had some trouble with the left ear; severe pain was followed by a discharge. After this she discovered that her hearing was defective in the left ear, and treatment made some improvement. The ear again discharged for three weeks in November, 1905. The last of December, 1905, she had sharp, shooting pains beginning in the back of the neck, radiating around the face to the right eye; aching pain constantly in right side of face and head. There was gradual loss of memory for six or eight months and a change in disposition, during which time there was an occasional nose bleed. Jan. 28, 1906, she had bleeding from the left nostril amounting to about four ounces; January 30, a profuse hemorrhage; February 1, slight bleeding; no discomfort or pain in the throat at any time.

Feb. 2, 1906, at the request of Dr. Williamson, Drs. Musson and Freeman examined the patient. A portion of a postnasal growth was removed for examination by Dr. Peckham, who diagnosed it an epithelioma.

February 7, Drs. Butt and Schneideman made the following report: Paralysis of the sixth nerve of right side, also of third and fourth, same side; lowering of sensation around right eye over distribution of the fifth ophthalmic division; fundus hyperemic; no optic neuritis; cupping of an oval disc, the long axis being 135 degrees; left eye fundus normal. They diagnosed probable basal lesion involving all the nerves passing through the sphenoidal fissure.

Through the courtesy of Dr. Williamson, to whom the case belonged and under whose care she was until the time of her death, I report the case. I examined the patient on February 4 and found in the nasopharynx a growth about the size of a large hickory nut, seemingly growing in two parts—one from the vault and the other from the left lateral wall. The lateral portion was in practically the same location as was the growth in the first case reported. The hearing in the right ear was not involved.

A few *x*-ray treatments had been given, when it was thought best to discontinue them, as they could not reach the seat of the trouble and might possibly do harm.

An operation was decided against and the growth gradually, but quite rapidly, increased until death occurred, Jan. 9, 1907.

This growth did not involve the right side externally, but it did the left to a great extent, although it did not go beyond the median line. Practically all the pain was on the right side of the head. There was no pain in the growth. The throat became pretty well filled by the extension of the growth from the left side, there being no involvement of the tissue of the right side of the throat. There was no pain in the throat; it only felt heavy and full.

There was total blindness in the right eye from June, 1906, with complete fixation of the eyeball and total blindness in the left eye for ten days in November, 1906, after which time she was able to distinguish light from dark. There was complete anesthesia of the right side of the face after July, 1906. This was preceded by pain in the dental nerves. In November there was slight pain in the left side, which was easily controlled. For from four to five months the patient could not chew or swallow anything solid, but took a good quantity of practically liquid food—a small amount of milk even the day before her death.

There was no marked loss of flesh or of appetite until the last three months, during which time flesh was lost rapidly. During six months before death there was a more or less severe hemorrhage every three or four weeks. These were promptly controlled by adrenalin, used on cotton through the nose, at the same time as much as a half-ounce being given in one dose by mouth, and two to four drops were given regularly several times daily. The half-ounce dose increased the heart action about ten beats. It was usually from 80 to 110 until she was obliged to stay in bed, the last two months, when it increased to 120. Two days before death it increased to from 140 to 160—finally not countable.

The last hemorrhage was during the ten days of total blindness in November, caused by getting out of bed alone. Up to the time of going to bed, she did many thing about the house. All symptoms were improved after each hemorrhage and grew steadily worse until the next. She never once complained of pain. Sleep was nearly continuous after November 2, but she could be roused and knew when spoken to. At Christmas she asked to have presents gotten for members of the family.

She had abundance of good, fresh air and all the food she could take. Medication consisted of strychnin and adrenalin. At intervals she had potassium iodid, gr. 10, increased to 40 or 60, three times a day. Mercury was given by mouth and by inunction, but was not well borne and was discontinued. Codein was used in sufficient quantity to prevent suffering, but never more than one-half of a grain at a time.

The patient lived until Jan. 9, 1907. This was about two and a half years after the first ear trouble, a little more than a year after pain in the head commenced and less than a year after the first hemorrhage—an occasional nosebleed having been noted before this.

Was there any time when a careful examination of the nasopharynx would have discovered the growth at a sufficiently early stage for an operation to have saved this woman's life? Would anything have been gained by removing the growth when first discovered?

DISCUSSION.

Dr. Stein:—I did not understand the doctor to say whether she knew what method of operation was instituted the second time the boy was operated on. The question arises in my mind whether it would not have been a very good idea to have employed the method of electrocauterization of the stump after the doctor had operated the first time and removed this specimen so beautifully. The method is pursued in cases of malignancy of the nose and retronasal fossæ by some men with considerable success. Price Brown of Canada, you know, has employed this method in the removal of large masses of malignant tissue from the region of the nose, particularly of the retronasal space, and has reported several cases. I have seen two of them operated on, and really it is encouraging to see the success this man has had by this method—enough to encourage one to employ this means. It is not new at all, and not considered by some of any particular value, but success even in the hands of a few should encourage us to employ it, and it seems to me it would have been a useful method in this case.

Dr. Beck:—The retronasal tumors are particularly interesting from the pathologic point of view, and it depends entirely upon the microscopic examination of the growth as to what the outcome will be, notwithstanding the failure of the microscope in Dr. Goldstein's case. Dr. Baldwin speaks of the findings of the pathologist in this growth. If you examine the tumor you will notice that it has a perfect outline; it has a pedicle and it has a sort of capsule which is clearly defined. Microscopic examination, endothelioma. True enough, endotheliomata have been classed as sarcomata, but we find that people who have an endothelioma sometimes live for a long time. It has been my experience in three cases, one of which was a primary endothelioma of the middle ear. Senn is plain in this point: if the endothelioma is extravascular, it is sarcoma; if it is a growth in the vessels, it is non-malignant, and this is very likely what the doctor had in that case. I believe that Bretsbaum's cases are exactly the same. This explains why so many of our nasal sarcoma cases live so long; the endothelial structures are within the vessels, and not outside. I think it is well to know that the pathology of these growths is determined only by the microscopic examination. Now, I wish to sound a warning. We see many cases where adrenalin is used. I think it is dangerous when long continued, for it does act on the vessels of the body. It causes an atheromatous condition prematurely, and there are dangers of secondary hemorrhage in these cases. Of course, in a case like this, anything might be used. *X*-rays are absolutely of no value in cases of retro-maxillary tumors or such growths.

Dr. McCaw:—I would simply like to emphasize the importance of diagnosis of which Dr. Beck has just spoken. I think it the most important factor. I would be somewhat suspicious regarding the diagnosis of this growth. It does not have the characteristics of a sarcoma. As Dr. Beck says, there is a pedicle and a distinct capsule. Rarely do we see such conditions in a sarcoma. Another thing, this growth was located at the choanal margin, high up, the position in which we usually expect to find transitional growths. That is to say, growths in the transitional stage between myxomata and fibromata. Another thing is the severe hemorrhage following operation is not characteristic of sarcoma; that is my experience. In considering the diagnosis of growths in this region we must not lose sight of the fibromata with a malignant clinical history, and often confounded with sarcoma. Frequently these growths will lead us astray in our diagnosis if made from the clinical history alone. In the second case the doctor speaks of, I have again to coincide with Dr. Beck. I am very suspicious of the diagnosis of epithelioma. I think it belongs to the class of carcinoma.

Dr. Voorhees, Elmira, N. Y.:—I want to say that I think it is of the utmost importance to have a careful examination made by a competent microscopist, and then we can not always depend upon what they tell us. Some time ago I had a case in point, a tumor of the nasopharynx. I brought a section to the microscopist who diagnosed it round-cell sarcoma. I know well enough, as Dr. Beck has said, there is nothing to be gained by using the x-ray in severe cases, but I tried it. There was no history to lead me to believe the case referred to might be specific, but I finally put the patient on specific treatment and she recovered. I supposed from the report of the microscopist it was a case of sarcoma. I believe with Dr. McCaw that the sarcomata do not frequently show hemorrhage in the early part of the growth, or even when breaking down or when they are able to be removed. And I would question the recurrence of that growth that Dr. Baldwin has shown. There might perhaps have been some mistake in the examination by the microscopist.

Dr. Faith, Chicago:—I want to ask one question and briefly relate the history of a case. A number of years ago I had a case of what appeared to be retropharyngeal abscess. There was a history of adenoids removed two or three years previously. The adenoids recurred and were removed a second time. This growth occupied a position beginning about the base of the tongue and extending well up into the nasopharynx, so that respiration was obstructed, making it very difficult to breathe, and there was also great difficulty in swallowing. The boy had a temperature of 102° and over, was very emaciated, and sweat profusely. Palpation led me to diagnose it as retropharyngeal abscess. I opened it, and the boy nearly bled to death. I could not get any pus. I cut out a section for diagnosis, which was made as sarcoma, and the boy never sufficiently recovered to undergo further operation. He afterwards saw Dr. Senn, who refused to operate, and he died in the course of four or five weeks. One point of importance in connection with the case was the temperature, which we later thought was due to inanition. I would ask Dr. Baldwin if any of her cases exhibited abnormal temperature?

Dr. C. L. Minor, Springfield, Ohio:—We are led to believe that malignant disease in the nose and nasopharynx is a rare occurrence if we are to judge from the amount of literature on the subject. In preparing case reports on this topic two years ago I was astonished at the few cases recorded and the very meager report of those few. If every one encountering these cases would report the same in full we would soon have a wealth of material from which to make deductions, for I am of the opinion that the disease is not so rare as we are taught. Again, in making these case reports, give every little detail. Let the pathologist give the microscopical findings on which he bases his opinion and not merely say it is a sarcoma, etc. If the essayist had given the pathologist's report we would not have had all this discussion as to what variety of tumor the doctor was presenting. Again, when an autopsy has been made give the report in detail, especially the microscopic examination of any suspicious nodes found in the viscera, for it is a much mooted question as to whether these tumors in the nose are primary or secondary. I should like to put in a good word for the microscopist, since it seems to be the disposition of this assemblage to criticise him for inaccurate work. He usually reports on just what is given him for examination without further to do, and we are disappointed that it does not bear out the clinical findings. Such failure is due to our own inability to secure the specimen for examination from the locality that we wish, and of course he finds nothing but normal structure, with possibly some necrosis, but no evidence of malignancy. In reference to the bleeding both during and after operation in the five cases of malignancy that I have had, the hemorrhage was terrific at the time of operation, and as a rule the patients did not live long enough afterward to make much observation on postoperative bleeding, although it is interesting to note that one case died some time after from hemorrhage from a secondary nodule in the intestine.

Dr. Ballenger:—I want to report a case here as fully as I can because I think it will clear up our ideas somewhat as to the diagnosis of sarcoma of the nasopharynx. I had a case which was seen by various laryngologists, all of whom are distinguished men—one of them was Dr. Goldstein of St. Louis, Dr. Gradle of Chicago, and Dr. Ingals of Chicago, and finally it fell into my hands, and while I do not know the exact opinion held by these men, I think they suspected it to be a malignant growth. The gentleman was 42 years of age, very large, strong, healthy-looking, when he came to me. He noticed the obstruction in his nose only about four weeks previously. After four weeks of subjective symptoms on the part of this patient he had a beginning enlargement of the glands on the right side of his neck. I transilluminated his face and found absolute darkness on the right side as to the antrum—every evidence of antrum disease. On examining the epipharynx I found a round swelling upon the right and lateral superior wall of the epipharynx. I told the patient that I suspected it was a malignant growth and said I would try to confirm the diagnosis by removing a piece or specimen with a large biting forceps. I sent the specimen to the Columbus Laboratories and their report was endothelioma, perhaps sarcoma. This specimen was sent to Dr. Fisch of St. Louis, and his report was that it was an endothelioma, with no further description. I then took the patient to Dr. J. B. Murphy of Chicago, and he, with his assistant, examined the specimen and reported it to be sarcoma, and advised non-operative interference. I then took the patient to Dr. Nicholas Seen, and later to Dr. Charles Mayo of Rochester, Minn., and they reported sarcoma. Dr. Mayo recommended the thing that I had been recommending to the patient, namely, ligation of both carotids and their branches; the object of this treatment, which is known as Dawbarn's method, is to starve the growth. Malignant growths require an excessive amount of blood to foster their growth, and hence by starving the tumor its growth would be checked. Of course, the lymphatics were already involved in this case. All we promised the gentleman was that we might give him a more pleasant mode of death at a later period in his life. The operation would relieve him somewhat, we told him, and the growth would probably go, by metastasis, to some other part of the body—the lungs, liver or spleen—and he would have a more pleasant mode of death. The patient asked us how long he would live without operation, and Dr. Mayo said three months; in case of operation we told him he might live one to two years. He returned to Chicago and decided to trust himself to Christian Science rather than the surgeon, and he died four months later. Now let me recall the description. This was *not* a pedunculated growth, but a *mass* on the right lateral wall of the epipharynx. Upon removal of a piece for examination there was no hemorrhage.

Dr. Goldstein:—The paper of Dr. Baldwin suggests several questions which are more or less difficult to deal with in this class of cases. I refer particularly to the bleeding, which is in the way of complete eradication. I recall a case under my care in which the diagnosis, about five weeks ago, from a section removed at that time, was of myxofibroma. Every time I attempt to introduce any form of instrument for removal of a specimen the bleeding is so profuse as to obscure the whole field of operation in spite of adrenalin or any other form of hemorrhage control that can be used. The question I want to emphasize is that in this class of cases, where frequent operations are undertaken at short intervals to gradually obliterate the growth, there is a danger of changing a comparatively non-malignant form of neoplasm into a malignant one. There is already a change going on in this particular case, as the microscope reveals. I think I have made some ten or twelve attempts in the last five weeks to get a specimen, and I am gradually working my way back to the nasopharynx, and still find a considerable mass. I am inclined to think that if I continue the operation, I will be able to report another case of malignant neoplasm. I do not think the point is emphasized enough. I think the matter of galvanocautery is one which will stimulate metamorphosis in the character of the neoplasm. The case Dr. Ballenger refers to is one I perhaps saw earlier than the gentlemen in Chicago. At that time it showed the location of the neoplasm in the right epipharynx, smaller than when Dr. Ballenger or Dr. Ingals had seen it. I only saw the patient once, but I suspected malignancy, and suggested the character of the tumor at that time, but he was scared off, and that is all I saw of the case. I am glad to hear details of the reported case.

Dr. Stucky:—You will recall some years ago when the Academy met at Indianapolis, I read a paper on a similar topic—Tumors in the Nasopharynx—and while I agree with all that has been said as to the necessity for accuracy in your diagnosis, yet that is frequently impossible. I believe that one reason why we get so many ill results, as described by Dr. Minor, and such ill results as so many of us are accustomed to, is that we do not do a clean operation on account of the terrific hemorrhage, which comes out with all the force and power that the cavities will permit. Remember in that paper I advocated the tying of the carotid artery, and I want to advocate that again in suspicious case of tumors that are not pedunculated and of which we are uncertain as to the report made by the pathologist from the little bite gotten from the specimen. It is safer, I believe, to tie the carotid, one or both, and I have a case now, which is under forced nourishment, in which I expect to tie both carotids and remove the growth. Only a few weeks ago I assisted Dr. Chevalier Jackson in an operation for malignant growth of the larynx in which the patient said he would rather take his chances for a year or two than suffer for months. We tied the external carotid on the left, and the common carotid low down on the right. I watched that patient very closely, his convalescence was rapid, and he walked out of the hospital on the tenth day, and a letter from Dr. Jackson, received a few days ago, says the man feels like a new creature. If the growth extends over the median line and involves the whole nasopharynx, I tie both carotids and give the patient and myself a clean chance.

Dr. Baldwin (closing):—I have merely to add that it was a very happy coincidence that I tried to remove this growth with the snare. Had I not done this, and so controlled the hemorrhage, I doubt if I should have been able to complete the operation. As to the pathologist's examination, this specimen was examined at the University of Pennsylvania and I think was given very careful consideration. The second case was examined by Dr. Peckham, in whose opinion I have very great confidence. I did not speak with Dr. Peckham about the case. I simply give the diagnosis as reported to me. As to the amount of adrenalin given in the one case, it was only given when there was a severe hemorrhage, and a half ounce promptly controlled the bleeding.

Dr. Stucky:—What was the immediate effect of that large dose of adrenalin? What was the effect of that enormous dose upon the heart?

Dr. Baldwin:—Simply raised the heart about ten beats.

PRIMARY SARCOMA OF THE NASOPHARYNX.

REPORT OF A CASE.

JAMES F. MCCAW, M.D.
WATERTOWN, N. Y.

The case I present for your consideration was seen Jan. 21, 1907. The following history was obtained:

J. W. M., aged 60 years, male, of Scotch parentage. Family history negative. Had been addicted to the use of whisky, going on periodical sprees for years. Has always been troubled slightly with "catarrh." For twenty years the hearing of left ear has been markedly impaired. The hearing of the right seemed good and noticed no difficulty with it until about one year ago, when it suddenly became impaired. This improved in a few days, but never returned to its original condition. These attacks of impaired hearing appeared at frequent intervals, leaving the hearing worse after each. For about six months has been able to hear only very loud conversation. About the time his right ear began to trouble him, he noticed slight obstruction of the right nostril, bloody discharge when clearing same, and blood-stained sputum when clearing the throat. This was followed shortly by a feeling of fulness and soreness in the throat, slight pains radiating over the entire right side of the head, and torticollis on the corresponding side. All the above symptoms increased gradually in severity, with loss of flesh and strength, and he was finally forced to abandon his employment. Several weeks before I was consulted he developed very severe intermittent pain in the right ear, worse at night, a noticeable difficulty in swallowing and muffled voice. Examination showed the following: his skin wrinkled and drawn, face bore an anxious look; from a man weighing 195 pounds normally he weighs now only 160 pounds. Temperature, 99.5° F.; pulse, 96. There was a crest-shaped deviation of the nasal septum to the right and a small polyp in the middle meatus on left side. In the nasopharynx was seen a tumor occupying the upper, posterior and right lateral wall, involving the right Eustachian eminence. It extended from the upper part of the nasopharynx down to a point opposite the margin of the soft palate, filling the entire right side of this space and in contact with the soft palate above. The surface of the tumor was smooth and firm to touch below; the upper part was breaking down and undergoing necrotic changes. This necrotic surface was covered with a foul bloody discharge. Although nasal obstruction was quite pronounced, neither the nasal chambers nor soft palate were involved in the growth, the obstruction being due to the presence of the mass in the nasopharynx. There was a muffled voice with nasal intonation and slight dysphagia. Based upon the classical symptoms, of the presence of a friable necrotic growth in the nasopharyngeal space, nasal and postnasal bleeding, severe pain in the ear and head on the corresponding side, stiffness in the muscles of the neck, foul postnasal discharge and well-marked cachexia, a clinical diagnosis of malignancy was made and a piece of the growth removed with forceps for microscopical examination. The pathologist's report revealed the fact that it was a round cell sarcoma. The question of diagnosis settled, treatment presented a most perplexing problem. Here was a tumor which had, in two weeks from the time I first saw it, continued to grow so rapidly that it now completely filled the postnasal space, forcing the soft palate well forward and downward, producing difficult and painful deglutition, some obstruction to breathing, quite severe hemorrhage from the nose and throat upon the slightest provocation, interference with rotation of the head and motion of the lower jaw, and pain so severe that large doses of morphin were required for relief. At this time the family and patient were willing to have anything done that offered hope of relief from his suffering.

The patient was taken to the City Hospital, and, after the ordinary preparation, chloroform was administered and a low tracheotomy done, a tube introduced and the chloroform continued through this. A mouth gag was now inserted, and, with the patient in the Trendelenberg position, the soft palate was divided in the median line up to the bony portion. The flaps were retracted, when the tumor, about the size of an ordinary lemon, could be plainly seen occupying the situation above described. This was removed as thoroughly as possible with forceps, scissors and curettes, but not to my satisfaction. It was found that there was no definite outline or capsule, but it was a diffuse growth, infiltrating the deep tissues of the posterior and right lateral walls of the pharynx. It was plainly evident that I did not and could not cut wide of the diseased area without injuring important structures. The hemorrhage being also very severe, I desisted from further interference. After checking the bleeding with iodoform gauze packing, the palate wound was closed with interrupted sutures. The gauze packing was removed in twenty-four hours, the tracheal tube on the third day and the sutures from the palate on the fifth day. In three weeks the patient left the hospital with the tracheal wound closed, great relief from suffering and dysphagia, nasal respiration free and voice clear. Postnasal examination at this time showed a small nodule just below the Eustachian tube. As the tumor was again increasing rapidly in size I attempted removing with forceps small pieces of the growth every second day, as suggested by Bosworth. This proved inefficient, and daily injections into the mass of 15 minims of adrenalin chlorid (1-1000) was resorted to. Immediate relief of pain followed each injection and lasted from one to two hours. The effect was almost instantaneous and thus far the writer has been unable to explain this phenomenon satisfactorily. The injection seemed to hold the growth in check more effectually than anything I had previously tried, but had to be discontinued on account of the rapid heart action and extreme dizziness which followed each injection. May 23 some enlargement of the cervical glands on the right side was detected for the first time, with pain and lacrimation of the right eye. The growth continued to enlarge rapidly and the pain became so intense that on June 23 the external carotid artery was ligated just above the bifurcation on each side. The superior thyroids were ligated separately. At this time the greater part of the growth was curetted away. Immediately after ligation of the external carotids, palpation of the tumor imparted a very soft, almost cystic feel. This was evidently due to the impeded arterial circulation to the part with a more or less collapse of the distended vessels, the venous side being still full.

Following this procedure the patient reacted nicely, the mucous membrane of the pharynx and nasopharynx became very pale, and there was immediate relief from the severe pain, torticollis and muffled voice. A very peculiar phenomenon was the fact that for three days following the operation the patient was unable to swallow liquids, but could take solids more easily. The wounds healed by primary union and the patient made an uneventful recovery, leaving the hospital seven days later. Weight, 128 pounds. For two months he improved rapidly, gaining twelve pounds in weight, complete relief from pain and discomfort of any kind, all hemorrhage and foul discharge ceased. Pulsation in the facial or temporal arteries could not be detected.

About this time the growth again began rapidly to increase in size, extending down the posterior and right lateral walls of the pharynx, crowding the soft palate well forward, interfering somewhat with deglutition, and pain again becoming quite severe. At this writing the case seems beyond hope of doing anything more than relieving his suffering with opiates.

The pathological report is as follows:

Character of Specimen.—Neoplasm from nasopharynx, soft, friable, of grayish-brown color.

Microscopic Appearance.—Slides show very little stroma, but a nearly homogeneous mass of round cells of connective tissue type with blood vessels among them, the cells being so deeply infiltrating and degenerated as to make the cutting of sections showing single layers of cells practically impossible. A few areas of spindle-shaped cells show, and some very large cells, almost simulating squamous cell carcinoma; however, the lack of stroma, position of blood vessels and great preponderance of small round cells seem to warrant the diagnosis of small round-cell sarcoma.—I. M. Meader.

Notwithstanding the number of cases of malignant disease reported, with the infinite variety of methods used in the treatment of the same, we are still far from the goal of our ambition in dealing with this most horrible condition. True it is we have made some advance in the treatment of this disease, but the results in general leave much to be desired. The general subject of malignancy should be of interest to us as physicians, and laryngologists should be specially interested in the subject of malignant disease of the upper respiratory tract. There is no subject in the domain of medicine of such importance, about which we know so little. Primary sarcoma of the nasopharynx is a rare condition. In reviewing the literature of the subject the writer has been able to collect only twenty-three undoubted cases of this disease, including the case herein reported. It might be mentioned that there are many reported cases of sarcoma of the tonsils, nasal passages and a few of the soft palate, involving the pharynx and postnasal space secondarily, but manifestly these could not be included in this paper, as the writer is reporting only sarcoma affecting the nasopharynx primarily. In looking up the literature of the subject the author has been impressed with the lack of exact data, in many reports making it almost impossible to classify the case as to its primary origin or pathologic nature. The writer would, therefore, emphasize the importance of more specific reports as to the nature and primary location of growths in this region, that our literature of the subject may be of more value. A brief review of reported cases is convincing evidence of the necessity for it.

Treatment opens a wide field for discussion; the writer will, however, allude only to those methods which have been found to be of value in dealing with this most dreadful malady. The injection of different substances into the tumor and into other parts of the body has of recent years been practiced quite widely in inoperable cases. Of these substances arsenic, formalin, alcohol, adrenalin, trypsin and amylopsin and the mixed toxins (Coley) have been most used and all have their advocates. So far as we have been able to find, Thompson of Cincinnati is the only one reporting a cure of sarcoma of the nasopharynx by any of the above injections. He used formalin. In view of the fact that his case was only under observation from April, 1899, to July, 1900, we would feel somewhat skeptical as to the ultimate result. It has been shown over and over again that malignant disease may recur or form metastatic deposits years after the disappearance of the original growth.

Chevalier Jackson reports a case with recurrence seven years after laryngectomy for carcinoma, and cites another where recurrence took place thirteen years after removal. From his experience he concludes there is no such thing as absolute cure of malignancy.

Trypsin and amylopsin have recently attracted much attention. While it is true trypsin may dissolve cancer cells and some patients improve under its hypodermic use, no cure has yet been reported, and the question of its beneficial action is still to be determined. Arsenic, alcohol and adrenalin have proven of some value in relieving suffering, but without any claim of curative effect. The Finsen light and *x*-ray are still under trial with all probabilities against their general usefulness. Their curative influence we think has been greatly overestimated. The advantage of the Roentgen ray being only in the fact that it exerts a beneficial analgesic effect and causes a diminution of offensive discharge, therefore, is best used after the excision of the growth.

With our present knowledge of malignant disease, surgery in some form seems to offer the greatest hope of success. The methods usually practiced are (*a*) removal piecemeal at different sittings, (*b*) removal en masse, (*c*) ligation of the internal carotid on each side, (*d*) ligation and excision of the external carotid, (*e*) radical external mutilating resections. So far as the writer is aware, Bosworth is the only one reporting an absolute cure with any of the above methods. He removed small pieces of the growth every second day and finally succeeded in preventing a recurrence. This subsidence of growth extended over a period of seven years. It seems difficult to account for the discrepancy in results obtained from the use of the same method in different hands. For example, the writer tried the method in his case by which Bosworth obtained such an excellent result and it proved entirely inefficient and was abandoned. It would seem therefore that there is something aside from the method employed that influences the result and that something must be inherent in the growth itself, the individual, or possibly both. Our present knowledge of the cause and nature of malignant disease is entirely inadequate to definitely explain these discrepancies. May we not have different degrees of malignancy inherent in growths, pathologically of the same type? The same, for example, as we have in different degrees of virulency in typhoid fever, diphtheria and many other conditions? This, together with the variable resisting power of different individuals, would, theoretically at least, account for the cases of reported cures of malignant disease. The whole question of malignancy is still so indefinite that we are unable to say the length of time that must elapse before a recurrence or metastasis is improbable. Until we have more exact knowledge of these important factors we think it wise not to report our cases of malignant disease as cured, but as having remained so long without recurrence or metastasis. On account of the close proximity of important structures to the nasopharynx an attempt to remove a malignant infiltrating growth en masse can be nothing more than palliative. By an early recognition and immediate and thorough extirpation we might hope for better results from this method. We would strongly advocate such a course if the case is seen sufficiently early, but, as we all know, these cases are usually seen too late; at a time when the growth is infiltrating the deeper structures, and under such circumstances it seems to the writer that ligation of the external carotids either with or without resection offers the best chance for relief of symptoms and comfort of the patient. The external mutilating operations with the resultant deformities, as practiced by the general surgeon, are mentioned only to be condemned. There can positively be no hope of cure when a case has reached a stage that would seem to warrant such a procedure, and the relief of the patient can be accomplished by less mutilation and immediate mortality. Following such operations the patients either die on the table or death follows shortly after. In view of these facts we feel that such operations are unjustifiable.

DISCUSSION.

Dr. M. F. Coomes, Louisville:—I listened to the paper with a great deal of interest and I want to report a case that is very interesting because of the length of time since the removal of the growth. In 1895 a man was brought to my office with a growth in his nose, and the man who brought him was rather urging and insistent that I remove the tumor in the office. I made a casual examination and saw a large tumor in one nostril, which I attempted to remove with a hook, and to my very great surprise I had a terrific hemorrhage, almost as much as if I had cut the carotid artery. I had to plug both anteriorly and posteriorly to stop the hemorrhage. I then submitted a portion of the growth to a microscopist of this city who pronounced it sarcoma. The man was under my care for a year and a half before I finally made a radical operation. Sections of the tumor were examined by several microscopists, all agreeing that the tumor was a round-cell sarcoma, and I was advised by everybody, and the dictates of my own conscience, to let the man alone; that he would live longer without the operation than with it, as after that first operation it grew so rapidly and enormously. Finally I concluded that I would try Coley fluid. It made the man desperately sick, an enormous slough followed, and I thought for several days the man would die. Finally the growth developed very rapidly and repeated microscopic examinations were made, all confirming the fact that it was sarcoma. The man came to my office and said: "I have a wife and two babies, and do not want to die. What would you do if you were in my place?" I told him that I would get rid of the tumor. He concluded to undergo an operation. I put the man on the table and made an incision down through the nose and lip so as to turn one-half of the face to one side, and the tumor was entirely removed. The operation required six minutes, after which I plugged the nares anteriorly and posteriorly, and I am glad to say he is still living. This case was published, and I think was reported in the *American Practitioner and News.*

DR. IGLAUER:—Concerning one point brought out by Dr. McCaw: He raised the question as to why the adrenalin produced an anesthetic effect, and it ocurred to me that possibly this was due to the chloretone used in these solutions (1 to 2 per cent.), which is analgesic in its action. The doctor also concluded that ligation was the best method and I should like to ask what he thinks of Dawbarn's method, who uses ligation of the external carotid, and then injects paraffin into the peripheral end of the artery.

DR. MCCAW (closing):—I congratulate Dr. Coomes on the outcome of his case. It is certainly unusual to see a case recover after such extensive involvement as the doctor describes, and especially a round cell sarcoma, for as we all know, this is one of the most malignant forms of cancer we have. In view of the fact that it did so promptly recover it would lead me to look with some doubt upon the accuracy of the diagnosis. Regarding the treatment of growths in this region, I am now strongly in favor of ligation of the external carotid on each side as the first step in operation and then attack through the mouth and remove as much of it as possible. This starvation method of treatment seems to offer the greatest hope of relief of any method we have at present. As I said in my paper, the prognosis in any case is grave and the results discouraging.

RETROPHARYNGEAL ABSCESS.

REPORT OF SOME UNUSUAL CASES.

M. A. GOLDSTEIN, M.D.

ST. LOUIS.

The recognition of retropharyngeal abscess or lymphadenitis, retropharyngealis suppurativa, as it has been proposed to classify it, is by no means of recent origin in medical literature. As early a writer as Galen apparently refers to this affection as of common occurrence in his experience. Morell Mackenzie[1] refers to the history of the subject as follows:

"The first notice of abscess in the pharyngo-esophageal region dates as far back as the second century of the Christian era, when Galen related a case which had occurred in his own experience and which terminated in spontaneous rupture. From his manner of alluding to the case, it would seem that he had seen several examples of the same kind, most of which had ended fatally. No mention of the complaint was made by any other writer, so far as I am aware, until the middle of the eighteenth century."

From this period until the early 70's of the nineteenth century retropharyngeal abscess is rarely mentioned. The most classic monograph on this subject was published by Bokai (Sr. and Jr.), who in an extensive experience at the Children's Hospital of Budapest collected over 500 cases of lymphadenitis retropharyngealis.

It is evident, therefore, that these cases are by no means rare in recent literature, but it is a notable fact that with all of the subject-matter at our command many of the important data have been but imperfectly developed. Neither in the monographs nor in our numerous text-books on pharyngology has the question of the etiology of this subject been definitely established. It is especially to be remarked that there is a great scarcity of case reports in American literature, and even in the more extensive monographs, as those of Bokai, Baginski, Henoch, Koplik and others, it is quite evident that most observations in this field have been made by pediatrists and not by laryngologists. The scarcity of these reports in laryngological literature, the obscurity of etiology and the limited subject-matter in the text-books prompt me to present a report of three unusual cases of retropharyngeal abscess, with such comments as have been suggested in the observation and care of these cases.

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[1] Manual of Diseases of the Throat and Nose, vol. ii, p. 57.

Case 1.—Baby S., male, aged 20 months, was brought to me two weeks after the onset of an acute streptococcic bilateral tonsillitis. The condition had been diagnosed as quinsy sore throat. The patient was treated by the family doctor, who, according to the statement of the parents, did not deem it necessary to confer with a laryngologist. During the course of the treatment the doctor had made two incisions on the right side and one on the left side, and I am constrained to add several directly into the tissue of the tonsil, to liberate pus from what he thought to be one of the natural sequelæ of quinsy, namely, peritonsillar abscess.

There is nothing especially worthy of mention to add to this case. I found the pharynx wall markedly bulging. The induration and bulging extended laterally to the tonsils, and these, also, were much enlarged. Externally, the neck appeared quite swollen, and a number of lymphatics could be distinctly outlined. Perhaps there may have been some difficulty in a differential diagnosis between peritonsillar abscess on the one hand and retropharyngeal abscess on the other in the early stages of development of this case.

I made a deep incision into the pharynx wall and liberated a large quantity of pus. The child made an uneventful recovery.

Case 2.—Baby W., male, aged 6 months, was referred to me by Dr. Zahorski and admitted to my hospital Jan. 9, 1907. Examination developed the following data: temperature, 99.4; pulse, 140; respiration, 38. Patient breathing heavily, extreme pallor with evidence of beginning cyanosis. The head was bent backward, the child seemingly breathing better in that position. The superficial and deeper lymphatic glands of the neck, the postauricular and sterno-cleido-mastoid areas on both sides were greatly enlarged, the tongue swollen, and the posterior wall of the pharynx showing distinct bulging, infiltration and edema. The child gave forth incessantly short, hoarse, weak cries, and attempted to maintain the position with the head thrown far back. It had been unable to nurse at the breast for over twenty-four hours. The patient had been seen by a laryngologist forty-eight hours previously in consultation with several general practitioners. An attempt at an examination of the pharynx had been made, and a portion of the posterior pharynx wall about one-half inch to the left of the median line and above the free margin of the soft palate had been excised with a narrow cutting or punch forceps and submitted for microscopic examination.

The report of the pathologist who examined this specimen within a few hours after its removal was embodied in a brief note to the attending physicians, the substance of which was as follows:

"My Dear Doctor:—The examination of tissue from the pharynx section referred to me to-day reveals distinct evidence of lymphosarcoma, 'and I am very sorry for the little patient.' "

On the strength of this microscopic diagnosis, together with the other clinical evidence determined on examination, the physicians in the case made a fatal prognosis and told the parents that nothing further could be done in the matter. It was at this stage of affairs that the patient was brought to my hospital.

Fifteen minutes after arrival the little patient was placed in a recumbent position on the operating table with the head overhanging the table, and an aspirating trocar plunged into the pharynx through the wound made by the excised section of the pharynx tissue, the trocar being carried well down until the surface of the vertebra could be felt. On withdrawal of the trocar, a single drop of pus was seen about the tip of the canula. I originally selected this site for penetrating the pharynx with the trocar, because of the wound previously made in examination of the pharynx. On noting the presence of even a small drop of pus about the tip of the trocar, I felt positive that more pus would be found. Both on digital examination and on inspection with reflected light there seemed perceptibly more bulging of the pharynx well forward on the right side than on the left, where thus far all examination measures had been carried on.

With a long, slender bistoury, held with its flat surface against the palmar surface of the index finger, and projecting about three-quarters of an inch beyond the top of my finger, I penetrated the right pharynx wall, cutting from below upwards and making an incision of about one inch in length. This was followed by a gush of pus, and two ounces of thick, greenish-yellow pus were liberated. To insure free drainage, I opened a pair of dressing forceps in this incision, spreading the tissues in all directions, also making firm pressure on the neck on either side. Several unsuccessful attempts at nursing were made. Four hours after leaving the operating room the patient took about three drams of mother's milk from a spoon. The throat was swabbed every hour with an antiseptic alkaline solution.

Within twenty-four hours the temperature dropped to normal, the pulse to 120, and the baby took the breast and nursed with ease. The patient was discharged on the tenth day. The infiltration of the lymphatic glands had all disappeared and the wound entirely healed.

Case 3.—Baby R., male, aged 9 months, referred to me by Dr. Friedman, and admitted to my hospital Dec. 27, 1906. Temperature, 100.6; pulse, 138; respiration, 32. The history of the patient's present illness was somewhat indefinite in detail and extended over a period of some two or three weeks. Treatment had been undertaken by various physicians for bronchitis, grippe, tuberculosis of the neck, syphilis, etc.

The first unusual feature presented in this case was the difficulty of examination. I was unable to make a direct inspection of the pharynx, for every time an attempt was made to depress or draw out the tongue or, in fact, every time the mouth was forced open the child began to choke. The neck on the left side was swollen, a number of lymphatic glands could be distinctly outlined and were intensely indurated; an occasional glimpse of the interior of the pharynx presented a swollen condition of the posterior and lateral walls together with that of the left tonsil. Dr. H. W. Loeb saw the case in consultation with me the following day, but no more definite data could be obtained, even after digital examination of the pharynx, because of the patient's tendency to choke. It was decided to postpone further examination until the next day. The throat was swabbed with adrenalin solution, and ichthyol applied to the side of the neck. The baby was beginning to nurse with some difficulty.

At 11:30 the night of December 29 I was called hastily to the hospital, as the child developed rapidly increasing cyanosis. An immediate tracheotomy was decided upon. I performed this operation under Schleich anesthesia, and it may be of practical value to describe some of the complications that presented themselves during this operation.

I used the Schleich anesthesia because of the rapidly developing precarious conditions of respiration and circulation. The difficulty and rapidity of respiration, the rapid, thready pulse and clammy skin were all contraindications to the use of general anesthesia. In fact, the tracheotomy might have been undertaken without any form of anesthesia, either general or local, because of the condition of extreme collapse of the patient.

I was prepared to find a small trachea in an infant of nine months, but the unusually small caliber of the trachea in this child, an infant physically well-developed in every other respect, took me by surprise. I had neither tube nor canula small enough to introduce into this infantile trachea, and my assistants patiently held open the edge of the tracheal wound with hooks and bent hairpins, while I racked my brains for a solution of the problem. During my visit abroad last season it was my privilege to see much of the splendid neck surgery of Mr. Henry T. Butlin, of London. Among the practical devices which I saw used by this resourceful surgeon was an adjustable metal flange, intended especially for use in combination with soft rubber tubing in similar emergencies. I cut a piece of No. 10 (?) velvet rubber catheter one and a half inches in length, adjusted this to the flange and placed it in position in the trachea. After several coughing paroxysms the patient began breathing through this improvised trachea canula, and it was maintained in position for forty-eight hours. On several occasions the tube was dislodged suddenly by paroxysms of coughing, but was immediately replaced.

An unusually excessive secretion of mucus about the tracheal wound and in the tube was noticed, due either to the presence and irritation of the rubber tubing in the trachea or to active inflammatory changes in the pharynx. We experienced much difficulty in keeping the tube free of mucus. At first we resorted to sterilized feathers which were introduced into the tube; but the coughing paroxysms which followed were so severe that the tube was dislodged at almost every such attempt. The caliber of the rubber tubing was so small that but little inspired air could be exhaled by the patient unless the tube was kept constantly clean. I had never been partial to the use of feathers as a means of cleansing the tracheal canula, and I was indebted to an ingenious suggestion of my assistant, Dr. W. M. C. Bryan, by means of which we succeeded in keeping the tube clean and pervious in a more aseptic manner. A piece of medium-sized tempered wire about six inches in length was brought into service as an applicator, looped at one end for a handle and provided at the distal end with a long, narrow loop and a tiny hook, about which absorbent cotton was tightly twisted. The purpose of the hook at the end of the applicator was to prevent the cotton from slipping off while in use. Several dozen of these applicators were prepared aseptically and proved a valuable and sufficient substitute for the time-honored feather. When they were once used, they were laid aside, the cotton burned off, and the wires again wrapped with cotton were ready for use.

Immediately following the tracheotomy and the insertion of the tube, the cyanotic symptoms disappeared. The temperature still fluctuated between 103 and 105 with the usual septic curve. The pulse varied from 170, twelve hours after the tracheotomy, to 140, one hour before the retropharyngeal operation, three days later. Respiration from 32 to 50. At 10 a. m. on the day of the tracheotomy the baby took the breast without much difficulty, and continued to nurse at intervals of three or four hours until shortly before the time of opening the retropharyngeal abscess, when it refused to nurse.

The patient was watched constantly, day and night, and especial stress was laid on maintaining the tube in position, as with every spell of coughing the soft rubber tube was in danger of being kinked and forced out of the tracheal wound. On the second day following the tracheotomy an aluminum canula was in readiness and was substituted for the rubber tube. The patient began at once to breathe more freely, the secretion of mucus was lessened in quantity, the tube was more readily cleansed and more easily maintained in place. The child's physical condition was now sufficiently improved to warrant a more exhaustive examination of the pharynx, and, while no distinct fluctuation in the pharynx wall could be obtained, the bulging, temperature and other clinical data all warranted the diagnosis of retropharyngeal abscess.

At noon on December 31, three and one-half days after the tracheotomy had been performed, I made a deep incision with a bistoury, guided by my finger, into the left posterior pharynx wall, and liberated about two ounces of pus, burrowed deeply under the pharynx muscles. I dilated the wound with my finger and an assistant made firm pressure on the neck inwards and under the ramus of the jaw. The head of the patient was held downwards and backwards to avoid the possibility of pus finding its way either into the trachea or the esophagus. The draining of the pus immediately relieved all tension and swelling in the neck and glands.

At 6 p. m. I made a similar incision of the pharynx wall to the right of the median line, again evacuating about two ounces of pus mixed with blood. At midnight the temperature had dropped to 99.8, pulse 140, respiration 36. The patient slept well nearly all night, waking at intervals of two hours, at which time the tube was cleansed, and the baby nursed. The aluminum canula was maintained in position until January 20, a period somewhat over three weeks.

In so young an infant, where the respiratory tract is of necessity unusually delicate, and where even slight irritations are very likely to produce unfavorable complications, I wish to emphasize the importance of a therapeutic measure, the value of which I was well able to observe, especially in this case. In many clinical reports of children under 1 year of age, where a tracheal tube has been maintained for a protracted period, fatal results have ensued because of septic bronchitis or pneumonia. In the compound tincture of benzoin we have a valuable agent to prevent sepsis in the lower breathing tract and to soothe the irritated and inflamed mucous membranes in the postoperative treatment of this class of cases.

In this particular case, compound tincture of benzoin was vaporized freely and constantly in the bedroom. I used a simplex vaporizer with a conical tip, and every few hours had the vaporizing apparatus brought to within two feet of the patient's throat and the vapor directed specially to the tracheal tube for ten or fifteen minutes at a time. Nor was this precaution of benzoin vaporization relinquished at any time during the conduct of the case. I insisted on the room being constantly saturated with this medication. This vapor was so penetrating that for several months after the patient had been discharged from the private room of the hospital the smell of benzoin was very apparent, though the room had been daily ventilated.

Tracheotomy has become so common as a surgical procedure that its technic and the simple conduct of the operation require but little comment. It is the after-treatment of these cases which frequently taxes the resourcefulness and ingenuity of the surgeon to their full limitations.

There was another condition associated with this case that may be worthy of comment. When the retropharyngeal wound had healed and all swelling and edema about the larynx had disappeared, there was no further cause for maintaining the tracheal canula in position. On withdrawal of the tube we found that another change had taken place. The child was unable to use its voice. When the tracheal opening was plugged up and the child made an effort to cry, there was a peculiar, hoarse, whistling sound as of air passing through a tube of narrow lumen, the upper part of which was rigid. I advanced the opinion, concurred in by my colleagues in the case, that this loss of voice was due to a deflection of the natural axis of the trachea and larynx. The intense infiltration of the tissues of the pharynx, the marked bulging of the musculature of the pharynx walls, the maintenance of the tracheal canula in a rigid position in an infantile trachea for a period of over three weeks, all contributed their share to throw the larynx or the chink of the glottis out of position in their relation to the axis of the trachea. The column of air in the trachea did not strike the vocal cords direct, but was impinged and deflected from the unnaturally curved walls of the trachea. I was making every preparation, on the second day following the withdrawal of the tube, to insert an intubation tube into the larynx, with the hope of gently forcing the trachea and larynx toward their normal axis, when slight vocal sounds were noticeable in the child's efforts at crying. These greatly improved, and by the time the tracheal wound had healed a perfectly normal vocal function was re-established.

I have selected the above three cases because they offer in their diagnosis, variations, clinical data, technic and postoperative treatment and sequelæ many valuable object-lessons which are but seldom and but briefly described in the literature of this field.

In Case 1 we meet with a condition that is more frequent than a cursory glance into this brief report might indicate. It is very likely that many cases diagnosed off-hand as quinsy, frequently followed by peritonsillar abscess, have in reality proven to be cases of retropharyngeal abscess. Perhaps it is difficult, in the early stages of infiltration about the fauces and the rapid induration about the soft palate, which is the typical picture in peritonsillar abscess, to sharply differentiate this condition from pus-formation posterior to the pharynx muscle. At times the pus focus is so deeply buried behind the muscles of the pharynx wall that it is practically impossible, even in the later stages of retropharyngeal abscess, to detect the presence of pus by palpation, for the pharynx muscle is so thick and so indurated that no fluctuation is perceptible. This was apparent in each of the three cases above cited.

From the literature of retropharyngeal abscess at our command, it is a noteworthy fact that nearly all cases which have been reported have been in children under 5 years of age. It occurs somewhat infrequently in older children and with extreme rarity in adults. If we exclude from our etiological consideration the small proportion of tubercular and syphilitic caries of the vertebræ, we must still account for the causative factor in the great majority of these cases. Why should retropharyngeal abscess occur so frequently, in the opinion of nearly all observers, in very young children, and why should this affection occur with increasing rarity the older the patient? We know that in early childhood there is an unusual activity of the lymphatic ring and of the lymphoid tissue in the pharynx, and that there are several small, deeply located lymphatic glands imbedded in the connective tissue of the posterior pharynx. It would be logical to assume, therefore, that whenever an acute infectious process takes place in the nose, nasopharynx, accessory sinuses, ear, tongue or larynx, a continuity of lymphoid tissue may readily carry such an infection into the depths of the pharynx walls. If the presence of this lymphoid tract in the pharynx areas is accountable for the frequency with which retropharyngeal abscess occurs in very young children, then the scarcity of its occurrence as the age of the subject advances may also be conversely proven, for, with adolescence and maturity, the lymphoid tissue which is so great a productive agency of hypertrophy or disease of any part of Waldeyer's ring greatly atrophies, and the pathology depending on this tissue is of necessity lessened.

Lymphadenitis retropharyngealis *without* pus formation is of rather frequent occurrence. The active stimulation of the lymphatics, the liberal distribution and wide radiation of the lymphatic glands in the pharynx itself and the susceptibility of young children to catarrhal inflammation of the mucosa of the upper respiratory tract substantiate the conclusions of such observers as Bokai, Baginski and Kormann, that in a large preponderance of patients so affected an enlargement of the postpharyngeal lymphatic glands could be determined. In the early nomenclature of this affection retropharyngeal lymphadenitis was classified either as idiopathic or as symptomatic. With the development of a clearer understanding and appreciation of the physiological and pathological importance of the lymphatic ring in its relation to affections of the pharynx, it is evident that a revision of this nomenclature could be made. While it may not be possible in each and every instance to definitely ascertain the first focus of infection, the culminating point of this affection may always be traced to an inflammatory or septic condition of the retropharyngeal lymph nodules.

In a subclassification, therefore, of this field, instead of referring to an idiopathic or symptomatic form, it may be more logical to consider retropharyngeal lymphadenitis either as inflammatory or suppurative.

Case 2 presents another valuable object-lesson. The clinical picture of this pharynx must have been very misleading, for it prompted the consultant in the case to consider the possibility of a malignant neoplasm and to remove a section of tissue from the bulging, indurated pharynx wall and submit it for microscopic examination. I can appreciate the hesitation of this laryngologist to offer a definite diagnosis without microscopic verification, for the tissue was dense, the swelling fairly circumscribed, and no other clinical data were apparent. The pathologist is the one who, in this case, might be open to criticism, not so much because of his error in the diagnosis of the microscopic section, but because of his positiveness in maintaining the diagnosis irrespective of any other data, and thereby misleading the clinicians engaged in the case. It is not difficult to understand how a diagnosis of lymphosarcoma could be made in the case under consideration. The induration and swelling of the pharynx muscle and the natural distension of the fibers of this tissue, together with the infiltration of lymph cells in the course of such an active inflammatory process, in a section taken parallel to the muscle fiber of the pharynx, might readily present a picture under the microscope similar to that of lymphosarcoma.

If the pathologist, however, were in closer touch with the clinician, and were informed of the clinical aspects of the case, of the induration and swelling that had taken place in the tissues, and the location and direction of the section which had been removed for microscopic examination, it is possible that such error might have been avoided. This experience simply emphasizes our position that, where a microscopic examination is called into question, it is a dangerous policy to offer a diagnosis based exclusively on such microscopic findings. The value of the microscope and of our pathological information can only be best developed when applied in conjunction with an intelligent understanding of the clinical data furnished in a given case.

The technical difficulties developed in the few cases which I have presented only serve to illustrate to what extent the ingenuity and mechanical skill of the operator may be taxed. The instrument catalogues present various forms of a combination bistoury and forceps for incising and draining retropharyngeal abscess, and yet there is nothing more serviceable than a slender, sharp, pointed bistoury, guarded and guided by the index finger of the operator.

A recent writer advocates that the patient, during operation for retropharyngeal abscess, be held in an upright position in the arms of an assistant, and the head well thrown back, a position similar to that frequently preferred for adenoid operation. This position of the patient seems illogical to me, especially where a large quantity of pus is likely to be liberated, for, as a large proportion of these cases consist of young children who are unable to assist in expectoration or to cooperate with the surgeon by following his directions, it is possible that some of the pus might find its way into the trachea, causing septic inflammation of the bronchi or lungs. To obviate this, I direct the assistant to hold the patient flat on the back with the head overhanging, so that the outflow of liberated pus may be easily controlled.

The tracheotomy undertaken as an emergency in Case 3 is a step which, fortunately, is not often necessary in this class of cases, for even under most favorable circumstances tracheotomy in such a young subject is a delicate and difficult technic, and the prognosis usually bad.

The incision of the pharynx wall should be free, deep and long, and the operator should observe the precaution to remain near the median line with the bistoury, so as to avoid wounding the internal carotid artery. Both in retropharyngeal and in peritonsillar abscess I make it a rule to introduce a pair of slender uterine dressing forceps into the incision and to spread the blades as wide as possible to break up any septa containing pus sacs and to enlarge the drainage area. In one of my cases a second incision was necessary in the opposite wall of the pharynx, and I attribute this to the fact that the drainage following the first incision was imperfect.

I have used the data of these recent cases to bring out some of the features of retropharyngeal abscess that have either not been mentioned or have been but briefly touched upon in the literature of the subject. With our increasing knowledge concerning the physiological and pathological importance of the lymphatic system and its relation to many of the diseased processes which we encounter as laryngologists, the subject of retropharyngeal abscess will, no doubt, be more prominently considered.

DISCUSSION.

Dr. Baldwin, Philadelphia:—I want to report one case of special interest—a retropharyngeal abscess which pointed and was opened external to the angle of the jaw. It came to the hospital late at night and was referred to a laryngologist who was in the hospital at the time. An immediate operation was necessary. The abscess was opened and packed with gauze. The next morning the case was referred to me. The child was not much more than alive. The packing was so tight as to cause the same symptoms as the abscess. By removing the packing you could pass a probe into the retropharyngeal wall without the slightest difficulty. It was interesting to me because it had been referred by a laryngologist. Free drainage was secured and twenty-four hours later the case was doing nicely.

DR. STEIN:—Was the incision made outside the neck?

DR. BALDWIN:—The incision was made outside.

DR. SPOHN, Elkhart, Ind.:—I enjoyed the paper very much, but never knew these cases were so rare. I have never looked up the whole history of these cases; but, because the cases were not considered of such great importance, many might not have been reported. I well remember a number of years ago, when I had a case of this kind in a little child three months old, and made a mistake in my diagnosis. The physician who referred the case to me told me the patient was dying. The child was so emaciated and could not nurse that I concluded that he was about right. Its temperature indicated that there was pus somewhere, but I did not think of pus in the postnasal region. As the child had adenoids, it was concluded that their removal would give the child more breathing space. The child was very dyspneic. In curetting the adenoids, I opened the pus site, which gave the child immediate relief of the dyspnea. The child made a complete recovery in four to five days. I have seen a number of cases in adults, but as stated before, I never knew that these cases were so rare.

DR. GOLDSTEIN:—I haven't much to add except, in looking up the literature of this subject, I concur with those gentlemen who have observed them so frequently, that retropharyngeal abscess is not rare in young children.

SOME OBSERVATIONS ON HYPERESTHETIC RHINITIS (HAY FEVER), WITH SUGGESTIONS FOR ITS RATIONAL TREATMENT.

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My observations on the study of hyperesthetic rhinitis, or so-called hay fever, lead me to the belief that this much misunderstood and mooted subject is not so intricate as many authors would have us believe.

It will be impossible at this time or in such a paper to undertake to discuss each and all of the various theories that have been advocated in the explanation of the classification of this disturbance. My remarks at present are but the expression of a few observations I have made from the study and treatment of a great number of such cases.

In order for you to understand my position as to the classification of this disorder, I wish to say at the outset that every synonym applied to this affection other than hyperesthetic rhinitis should be rejected on the grounds that they are misleading and confusing, and because the latter more correctly states the true condition present.

The writer believes this disorder represents a hyperesthesia of the trigeminal nerves, created by the instability of the body fluids and manifesting itself as a rhinitis with various reflex phenomena.

There is some internal factor at work undermining and lessening the stability of the nerve tissue, and that factor I believe to reside in the fluids of the body. This thought has given birth to the many opinions expressed by different observers and investigators in their uric acid theory, intestinal toxic theory, anemia theory, saliva theory, etc. What is called the idiosyncrasy of the patient is nothing but a perversion in the metabolism whereby some one or several of the fluids of the body become sufficiently changed from their relatively normal composition, so that whenever nerve tissue comes under its influence, becomes susceptible to irritation. Thus is furnished the first element necessary for the manifestation of the disease. The common belief that this hyperesthetic condition is created by a general neurotic temperament becomes untenable from my viewpoint. Many authors maintain that such a condition is absolutely essential to its origin. But I am not alone in having observed cases that were phlegmatic in all but their hyperesthetic fifth nerve or some other special nerve tissue. Furthermore, the possibilities of developing a general neurotic disturbance as a consequence of a persistent and recurring hyperesthetic rhinitis becomes evident to many physicians who themselves suffer from this malady. So I do not hesitate in saying that the general neurotic manifestation, so evident in many of these sufferers, is the offspring and not the parent of this disorder.

With the susceptibility established, the second element in the form of an external excitant, like dust, pollen, a draught, bright light, cold pressure spots within the nose, etc., readily complete the circuit, and the disorder is manifest. Eliminate either one of these elements and the syndrome of symptoms so characteristic of this affection is prevented.

The second or exciting element must not necessarily enter the nostrils to set up an irritation. Any place where there are branches and terminal filaments of the great fifth nerve may be the sensitive area. A bright ray of light entering the eye may irritate an individual with hyperesthetic ciliary nerve filaments and precipitate reflex symptoms in the nose. A draft of air from an open window or door, or from an electric fan, may irritate the terminal filaments that go to the side or back of the head, and there immediately results these same reflex symptoms. In another individual some of the terminal branches of this nerve within the nose constitute the hyperesthetic area, and an irritant applied there, like a septal ridge, dust or pollen, calls forth symptoms of the disorder. Owing to the nose furnishing the greatest area for the distribution of the filaments of the first two trunks of the trigeminal nerve, it is here we most commonly find areas of irritation.

If in a patient subject to this disorder we subject that particular branch or filament of his fifth nerve to irritation, we produce experimentally all the symptoms of the disorder, irrespective of place or season. I have already shown this in the experiments made in 1904 with the toxin of rye pollen, and reported in a communication read before the Chicago Medical Society, Feb. 22, 1905.[1]

Aschenbrandt, in 1885, showed by his experiments the susceptibility of the fifth nerve to irritation. He exposed in animals the spheno-palatine ganglion, and by electrical stimulation observed pronounced swelling of the cavernous tissues of the turbinals, with hypersecretion.

In the treatment of these cases our first thought must be directed toward the cause of the patient's susceptibility. From my prefatory remarks it will be seen that this cause resides within the fluids of the body, hence our search for the particular condition that has brought about the individual's susceptibility must necessarily partake of both physical and laboratory examinations. The condition of the gastrointestinal tract, with its dependent organs, must be investigated; the blood and urine examined, not only once, but several times. As this all demands considerable time, this information should be obtained somewhat in advance of the usual periods of attacks. Having thus before us an average analysis of the patient's condition, we may direct our treatment intelligently in the direction for the correction of any gross deflection from the normal. In some cases the acidosis will demand an antacid, like carbonate or lithium or bicarbonate of sodium. Faulty assimilation and elimination must be corrected by a regulated diet, proper hygienic surroundings and a sufficient amount of exercise in the open, as well as by the aid of a few well chosen drugs. As an eliminant I consider sal-hepatica both agreeable and efficient. It is claimed to contain, in addition to the salts found in the European "bitter waters," lithia and phosphate of sodium. Bishop's practice of administering acids early in the morning and at night, in order to lessen the alkalinity of the blood, thereby freeing it from uric acid, is good practice and of benefit in those cases where the nerve susceptibility is caused by an excess or the retention of uric acid. During an attack I have secured the most satisfactory results from the employment of a tablet of morphin and atropin. Bishop's formula contains 1/12 gr. of the former and 1/600 gr. of the latter drug, in addition to 1/6 gr. caffein.

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[1] Published in Medicine, April, 1905.

The second part of the treatment is concerned in eliminating the external irritating element or prohibiting its perception. As already pointed out, the causes at work here vary even more than those concerned in the predisposition.

The exclusion of the irritant, if it be dust, pollen, etc., by the use of nasal filters or change of habits, is beneficial in selected cases. The correction of irritating intranasal causes should be undertaken where indicated. The topical application to the nasal mucous membrane of remedies capable of relieving the unusual sensitiveness of its nerves is admissible when properly selected and applied. There are two classes of remedies used here, those producing a hyperirritating or caustic effect, like formalin, chromic acid, chloracetic acid and electro-cautery, and those producing an anesthetic and ischemic effect, like cocain and adrenalin preparations, and intraneural injections. It is of this latter method I wish to speak particularly.

Intraneural injections have already been used for the purpose of anesthetizing a field for operation, and they have also been employed in the treatment of neuralgia; but, as far as I am aware, they have never been employed in the treatment of hyperesthetic rhinitis until I made my first injection about a year ago. I have used alcohol for this purpose, following the suggestions of Schlösser in the treatment of neuralgia.

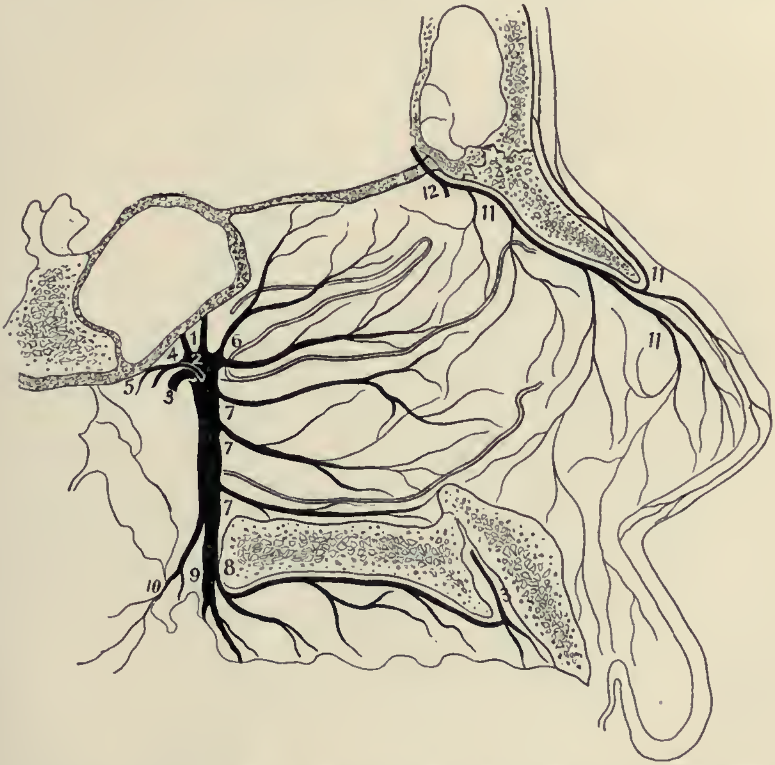

Fig. 1.—Spheno-palatine nerves. 2. Spheno-palatine ganglion. 3. Naso-palatine nerve. 4. Vidian nerve. 5. Sphenoidal branch. 6. Superior nasal nerves. 7. Inferior nasal nerves. 8. Anterior palatine nerve. 9. Middle palatine nerve. 10. Posterior palatine nerve. 11. External branch of nasal nerve. 12. Septal branch of nasal nerve.

In order to prevent the action of most of the external irritating elements, it is essential to the success of this form of treatment to inject the nerves as near their central ends as is consistent with safety and accessibility. This can be done in most cases within the nose. By referring briefly to the distribution of the fifth nerve and by the aid of the accompanying drawings (Figs. 1 and 2) the various avenues open to irritation and their approach for the purpose of injection can more readily be understood.

The nose and its sinuses are supplied with sensation through the medium of the great fifth or trigeminal nerve, or, more specifically speaking, through its first and second trunks. This nerve supply is best grouped into an anterior and a posterior group. The anterior group is represented by the nasal nerve, a branch from the first trunk of the fifth nerve, or the ophthalmic. This branch arises from the ophthalmic trunk just at the entrance of the sphenoidal fissure. It passes from the outer side of the orbit beneath the external rectus, diagonally forward and across this cavity to the inner wall beneath the superior oblique, where it enters a small opening in the os planum known as the anterior ethmoidal foramen, and is thus in the ethmoidal labyrinth, supplying this area as well as the frontal sinus by ethmoidal branches. In leaving the territory of the ethmoidal cells it passes upward and again enters the cranial cavity at the outer side of the cribriform plate, running forward

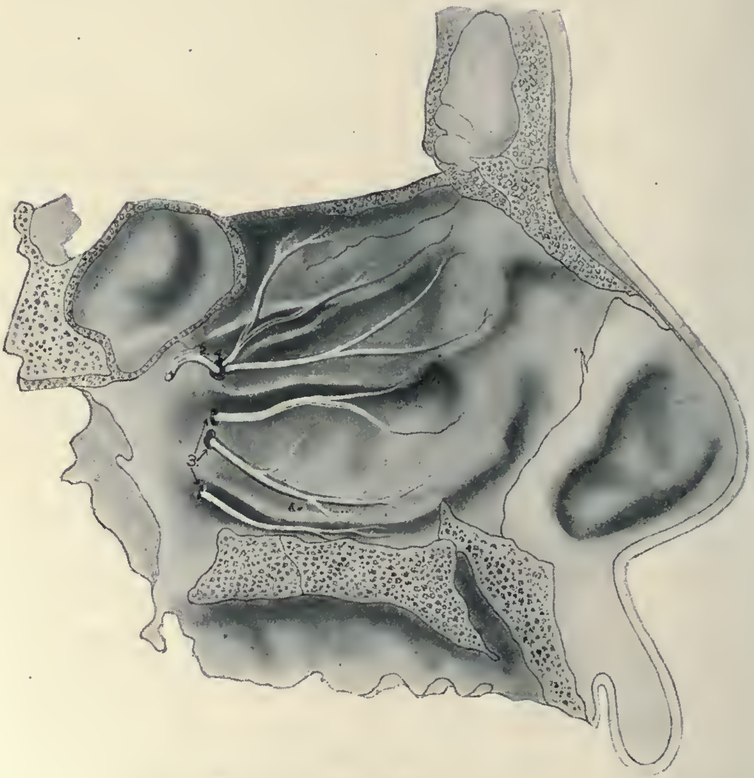

Fig. 2.—1. Spheno-palatine foramen. 2. Naso-palatine nerve. 3. Inferior nasal nerves. 4. Superior nasal nerves.

to the side and base of the crista galli, where it enters one of the anterior cribriform perforations and passes down into the nasal cavity. Its point of entrance into this cavity is at the extreme anterior-superior angle, dividing here into a septal and an external branch. The septal branch passes downward and forward over the anterior part of the septum. its filaments supplying the anterior portion of the perpendicular plate and quadrangular cartilage. The external branch passes downward in a groove on the under side of the nasal bone, its various filaments supplying the anterior part of the middle and lower turbinal with their neighboring wall and the nasal vestibule. The terminal filaments of this branch leave the interior of the nose between the end of the nasal bone and the superior lateral cartilage, supplying sensation to the ala, tip and bridge of the nose and also the area of the eyebrow and frontal sinus.

The posterior group arises from the spheno-palatine ganglion, and consists of four sets of nerves. The first is the *naso-palatine nerve,* arising directly from the ganglion and passing through the spheno-palatine foramen, with the other of the posterior group, inward and downward, crossing the anterior surface of the sphenoid sinus, just below its ostium, and on the septum, then obliquely downward and forward in a groove on the surface of the vomer just below its upper edge, it enters the anterior palatine foramen and then through a canal situated in the intermaxillary suture, distributing its terminal filaments to the gum and palate.

The second set are the *superior nasal branches,* which, arising directly from the ganglion, pass through the spheno-palatine foramen into the upper and back part of the nasal cavity. They then pass forward to the mucous membrane of the spheno-ethmoidal fossa. the superior and middle turbinals and a few filaments to the posterior ethmoidal cells and maxillary antrum.

The third set are the *inferior nasal branches,* which are offshoots from the anterior palatine branch of the ganglion. They pass from the palatine canal through openings in the palate bone, and supply the middle and lower meati and lower turbinal.

The fourth set is the *sphenoidal branch,* arising from the vidian shortly after its origin from the ganglion, and supplies sensation to the sphenoid sinus.

In addition to the superior nasal branches, the maxillary antrum is supplied by branches from the posterior-dental, which comes from the superior-maxillary root after the formation of the ganglion.

Within the orbit the nasal nerve gives off several small branches, one going to the ciliary ganglion, called the ganglionic; two or three supplying the ciliary muscle and iris, called the long ciliary, and one to the inner angle of the eye and side of the nose, called the infra-trochlear.

The frontal nerve or main branch of the ophthalmic root is known toward its terminus as the supraorbital, being distributed to the upper eyelid, brow, forehead and parietal region.

The lacrimal branch of the ophthalmic supplies its gland and also the upper eyelid.

These latter nerves are mentioned in order to show the wide field subject to reflex disturbances.

That the branches of these two great nerve roots may represent the hypersensitive area seems to be shown in the relief afforded certain patients after intranasal operation and treatment. But in the removal of a spur or the cauterization of a turbinal, or after irrigation of a sinus, only a few of the many nerve filaments within the nose are reached, and that is why the results following many such operations prove of but partial or no benefit. If in addition attention were given to the entrance of the nose, through which many irritating causes find their way, no doubt many more cases would be relieved. Upon this principle is based the value of the small nasal filter, consisting of a piece of fine mesh fabric enclosed in a metal frame and inserted just within the nostrils, thereby filtering the inspired air of almost all irritating matter, although it will not keep out irritating vapors, gases or odors, which may be the cause of precipitating an attack as readily as dust or pollen. But the irritating cause may be directed to the external terminal filaments of the nasal nerve which go to the forehead, brow and exterior of the nose, and in order to relieve these filaments of their hypersensibility something more than filters, cauterization of the turbinals, the removal of ridges and the like becomes necessary. By injecting the nasal nerve at its point of entrance into the nose, we deprive both its internal and external filaments of their hypersensibility. If we are to afford immunity to all cases of this disorder, it will be necessary to inhibit the action of the entire nerve supply of the nose, which means anesthetizing both the anterior and posterior group of nerves. This is easily done by selecting the proper length and curve of hypodermic needle and introducing it into the neighborhood of these nerves as they enter the nose. In the case of the nasal nerve, a needle two inches long is required, whose point is entered at the antero-superior angle of the nasal cavity, against the inner surface of the nasal bone, and near its distal end. The posterior group of nerves is reached by a needle three inches long with a curve near its end of 45 degrees. This is entered in the neighborhood of the spheno-palatine foramen, which is located in the spheno-ethmoidal fossa, just above and to the outer side of the posterior end of the middle turbinal. Both nostrils are injected, requiring four injections, two for the anterior and two for the posterior groups. In some cases it may be necessary to inject only the anterior groups. Both dilute and pure alcohol has been used by me for injection; using only a few drops in each injection.

The injections are made painless by a preliminary spray of a 3 per cent. cocain solution. No serious ill results have been encountered. I have found that the injections had to be repeated from one to four times, covering the season of attack.

It would appear from a study of the distribution of the fifth nerve that, in a few cases, we might still fall short of depriving all of its filaments of their hypersensibility, unless we made our injections central to the offshoot of the ethmoidal branches. These branches supply the anterior ethmoidal cells and the frontal sinus, and hence the nasal nerve would have to be reached from within the orbit, just as it enters the ethmoidal foramen. Experiments on the cadaver show this to be an easy procedure by means of a fine hypodermic needle two inches long introduced at the upper angle of the orbit and carefully hugging the bone.

As yet the work carried out by me along these lines has been mainly experimental, and a sufficient number of cases have not been under observation long enough to make this report more than preliminary. But I think with the aid of other workers along this line a most rational mode of treatment may ultimately result from this procedure.

DISCUSSION.

Dr. Spohn:—What the last two speakers have said with reference to operations upon the sinuses has been the experience of many rhinologists. I have had a number of cases upon whom I operated for sinus troubles, that obtained relief of the hay fever. It so happened that this was the exciting cause, which accounts for the relief. While this may relieve a few, it can not be made a universal rule. The only rule is the relief of the exciting cause. I know of a case that was refracted, and never had hay fever while she wore her glasses. Another case had an operation for appendicitis, and never had any hay fever afterward. It has been my experience that the old cases, say of fifteen or twenty years' standing, seldom obtain relief except by change of climate. Dr. Stein's method of handling is new to me; perhaps it is all right, but I believe it to be only temporary. I can not understand how the injection of alcohol can be curative, any more than the injection of water. The suggestions by the essayist upon the general system are good. But I would begin from four to eight weeks before the attack of hay fever. I believe the majority of the cases can obtain relief with a systematic general treatment. The organs of elimination should do their whole duty, the system should be in the best possible condition, and then with a properly restricted diet, the hay fever, if not entirely aborted, will at least be very much less in severity.

Dr. Beck, Chicago:—While I am not able to discuss this paper from any practical work done in this line, that is, on hyperesthetic rhinitis, I can speak on the use of alcohol injections in trifacial neuralga, having treated three cases in connection with Dr. Patrick at the Cook County Hospital for the relief of such conditions, following the experiments of several authors abroad on the injection of the fifth nerve at the exits of the foramen rotundum and foramen ovale. The technic applied by the doctor seems certainly very much in place in order to destroy the hypersensitiveness of the nerves that are involved in hyperesthetic rhinitis. I would say that in advance of all this work I think we are indebted to Dr. Cryle for the blocking system for anesthesia such as Dr. Killian, recently visiting in this country, has shown us. But Dr. Stein's work in this line is entirely new to all of us, I am sure, and to me it appeals very much. I would like to ask the doctor a question: Does he not think there might be some danger of puncture of blood vessels in the foramen, as we have some very large blood vessels there, and a hematoma might not be without some untoward symptoms? The injection in the region of the foramen ovale and rotundum is absolutely without danger, so far as large blood vessels are concerned.

Dr. Ballenger, Chicago:—I have been interested in Dr. Stein's presentation of this subject. It is the most suggestive I have ever heard. Back of all this subject is the question of what it is that renders the terminal filaments of the nerves hypersensitive. I want to speak of just one phase of it, to which Dr. Stein has alluded, that is, the influence of discharges from diseased conditions of the sinuses. Dr. Shadle some few years ago, and more recently also, has called attention to the influence of maxillary sinuitis upon hay fever, or hyperesthetic rhinitis, and I had arrived at a similar conclusion in reference to the ethmoidal and sphenoidal sinuses. I have found a few cases coming under my observation with this trouble that were relieved, not only at the time, but for as much as two years. One case to which I wish to particularly refer had the asthma and rhinitis so severely that she was compelled during July, August and September to sit up at nights in a chair and sleep with her head upon a pillow. After removing the ethmoidal labyrinth and some months later doing a double Killian operation, I gave her complete relief. In other cases I have had similar results, though not so striking as this one, hence it occurred to me during the reading of Dr. Stein's paper that possibly an explanation of these particular cases was that the hypersensitiveness of the terminal nerve filaments was due to the diseased secretions coming from these sinuses, and that in a certain percentage of the cases inflammation of the sinuses is, perhaps, the primary etiological factor in hay fever. Further than this I cannot discuss the paper on its merits because I have had no personal experience with his method of treatment, though it seems applicable to the relief of the paroxysms, if not to the cure of the disease. Then I want to raise the question as to the use of the term "distal." He, perhaps, means proximal. In the use of the hypodermic syringe, for instance, he used the word "distal," where I really believe he meant proximal. Am I right?

Dr. Dean:—I want to mention that I have operated upon four cases of sinus trouble that were suffering from asthma, with great improvement. Two cases required radical operations on both the frontal and maxillary sinuses, and in the other two simply the maxillary sinuses were operated upon. All of these operations were performed over a year ago; three of the patients consider themselves greatly relieved, while the fourth case is having now just as much trouble as before. In one of these patients much relief was secured before the operation by repeated irrigations of the antrum.

Dr. Holinger, Chicago:—There is an explanation which is very interesting and clears up some points which have been brought forth. Hay fever, just like bronchial asthma, is a reflex neurosis. The nervous element in this disease has often been shown. There are reflex-arcs which sometimes are very complicated. Wherever you take out one of the links you interrupt the arc, the reflex stops. As soon as the chain, through the healing of that interference of taking out the link, is closed again, you have the reflex arc closed. This explains the improvement from distant operative interference as in appendicitis, or antrum disease, or faulty nutrition of the whole body, etc. One indication is under all circumstances to lower the reflex irritability of the whole organism, and especially in the nose. One patient has regularly for the whole year washed his nose with a weak salt solution, and while for the last twenty-five years he had hay fever every summer, at the time when his day came—the first of August—and this year he was entirely free.

Dr. Willis, Indianapolis:—A couple of years ago, at Buffalo, Dr. Stein, and, I think, Dr. Beck, also gave us some very flattering results from the use of formalin in the treatment of hay fever. I wondered when Dr. Stein said he had been using alcohol in the treatment of cases of hay fever—I suppose he means ethyl alcohol—if he had found any relation between the action of this alcohol and formalin, as we know the chemical relation between methyl alcohol and formalin is so close, and the action of the various alcohols are more or less similar.

Dr. Andrews, Chicago:—There are, no doubt, a great many things to consider in this question of hay fever. In my remarks to the postgraduate students I am in the habit of stating that hay fever stands upon a tripod—that one of the legs is a constitutional tendency, another a local pathology, and the third an external irritant. I also tell them that after a person has had hay fever a sufficient length of time, it acquires an ability to stand upon two legs; that the local pathology may be corrected, and they will still have hay fever; that the external irritant may be gotten rid of by going somewhere, but they will still have hay fever. The constitutional tendency is extremely difficult to locate or eradicate. I think there is a great deal more in the constitutional tendency, so far as etiology is concerned, than we have been in the habit of considering. Professor Killian tells us of the hypersensitive areas in the nose, and that cauterizing them will relieve hay fever and prevent the attacks coming on. It seems to me that Dr. Stein has gone a little farther and anesthetizes the nerve before it reaches these hyperesthetic areas. I believe this is excellent work, and will bear further investigation.

Dr. Stein (closing):—In the introduction to my paper I observed that my remarks were only desultory and they do not necessarily stand for anything infallible. I observed that I arrived at these conclusions from the treatment of my own cases. The end of my paper relative to the treatment by means of injecting the nerve really demands an apology because it is not backed up by sufficient evidence. That particular phase of the subject is really not ripe enough to present, but it is pertinent to the subject that I thought I might presume upon your time to mention it and tell you what results I have had—and they have all been very satisfactory. I did not mention how many cases, because I have not counted them, but they are not very many; nor have I met with any ill results. As to the inquiry of Dr. Beck with reference to injury of blood vessels in the neighborhood of the posterior group of nerves, I will say that although it is true we have the large branches of the internal maxillary that supply the nose coming through the same foramen, but for some reason or other I have never had any ill results in injecting at this particular point. Maybe others will. I have asked a great many men to try this method of treatment wherever they had a chance. What results they have had I must yet hear. It is possible that somebody may ask: won't you have a great deal of pain from neuritis following these injections? Well, it seems possible to have a neuritis, but I have never observed any in my cases, and inasmuch as the injection treatment is used for neuralgia, I don't really see any objection to my treatment for that reason. I understand now, Dr. Ballenger's remarks as to the "proximal" and "distal" end, as I used it in the paper. I had in mind the terms as used anatomically, not in the neurological sense. I meant from the patient's standpoint, distal or central to the origin of the nerve. In speaking of the nasal nerve as it enters the anterior ethmoidal foramen, there are filaments given off in the ethmoidal labyrinth which go to the anterior ethmoidal cells and frontal sinus. Now, if we inject the nerve inside the nose where the nasal nerve comes through the anterior perforation of the cribriform plate, we would not reach the nerves in the frontal sinus. If the external cause is in the anterior ethmoidal cells, we probably would not get any results in such cases. Dr. Spohn referred to the treatment affording possibly only temporary relief. I still can only repeat that it is uncertain for what length of time they are relieved in these cases. So far my experience has been that where I inject them, they only require two or three, sometimes four, injections, taking only a few minutes at a time to do it. Whether they will find it necessary to come back next year, or not, we do not know. Perhaps they will. As to Dr. Willis' remarks as to whether the use of alcohol was suggested by my use of formalin, I will say no. In the paper referred to by Dr. Willis I made no claims to the use of formalin in the treatment of hay fever, but I mentioned Dr. Ballenger on the treatment of hyperesthetic rhinitis by means of formalin. I use the ethyl variety of alcohol in the treatment. Dr. Andrews believes that where you remove the external irritating factor, like dust, pollen, etc., you may still have manifestations of the disease. Well, I think that is simply because you have not removed the particular external irritating factor. You are presuming it is something that can be changed by climate, but that is not true in all cases, and to circumvent all forms of external irritants I get at the nerve that is irritated by injecting it.

SOME POINTS IN THE FUNCTIONAL DIAGNOSIS OF DISEASES OF THE EAR.

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It is the intention of this paper to clear up a number of points which have been brought forth either in literature, in discussions or in private conversation concerning the diagnosis of invisible diseases of the ear from the changes of function of the organ.

The first point is that cases with typical finding of changes of function are said to be very exceptional, and that the examinations take too much time. To refute this point Dr. Ray, of Louisville, was asked on very short notice to gather some cases of impaired hearing. He had five cases. All five were examined, taking the history, etc., in less than an hour. There is no need in entering into a description of each case; suffice it to say that all five were typical, namely, one case of occlusion of the Eustachian tube, one case of otosclerosis, one case of nerve-deafness from old age, one case of nerve-deafness in connection with syphilis, and one case of nerve-deafness in connection with a very old suppuration of the middle-ear. The patients were all present at the meeting. It is therefore evident, and it is the experience of all who have used functional tests for some time, that the cases are always typical and that atypical findings are a great rarity.

There are a few more reasons why functional tests are not generally used. They are:

1. Unless a man makes himself familiar with Bezold's train of thought before entering into the study of conflicting theories he will become confused and hopelessly mix up the different essential points. The clear logical consequences supporting the diagnosis are then absolutely lost.

2. A number of the most important papers appeared in the *Zeitschrift fur Ohrenheilk.*, of which the *Archives of Otology* is the American edition. This edition had short abstracts of some important papers and hardly conveyed to the American reader an idea of the value of the original investigation they contained. To make matters worse some of the German originals could be understood only with great difficulty by those who were not absolutely master of that language.

3. There is some very strong criticism and opposition in Ger-

many itself which has long ago made its way across the Atlantic and here found an expression in the words of a well-known professor of diseases of the eye and ear in one of the first colleges in Chicago, who said, "Well, whatever Professor so-and-so says is good enough for me."

Against this I say that the large number of favorable results of therapy based upon this means of diagnosis can not be shown by the other methods and are absolutely impossible by them. Laborious microscopical researches and investigations of pathology and of late even experimental pathology have completely confirmed all the main points in Bezold's teachings. The general acceptance by the profession of these teachings will mean a great boon to some of those most unhappy human beings who, on account of their impaired hearing, are excluded from normal social intercourse by means of speech.

A more careful study of the three points just mentioned might repay for the time given to it:

First.—There are any number of colleagues who have perseveringly tried to obtain a judgment of their own. It is very hard to argue with them, because the matter in itself is difficult.

In order to do away with the second reason against the general acceptance of Bezold's teachings in this country I have translated Bezold-Siebenmann's text-book of otology. It will appear by the end of December.

To explain the third reason why Bezold's teachings are not generally accepted in this country requires more time and the unearthing of a number of personal animosities which it seems to me ought not to have any weight with American scientists. The question is, will it have weight in the future? Nothing is easier for me than to show the unscientific motives and the lack of logic in this opposition.

Some of the points were illustrated to the members of the meeting in Louisville. In conclusion I only state that in all cases, where a functional examination was made and a diagnosis based upon it, the pathological finding after death coincided with this diagnosis. To demonstrate this fact a number of microscopical specimens from cases described in vol. liii, *Zeitschrift für Ohrenhielk.*, were at hand, but on account of lack of time could not be presented.

DISCUSSION.

Dr. Beck:—Those of you who are not familiar with Dr. Holinger, or those of you who are not as familiar with him, or as well acquainted with him as I am, would not grasp the meaning of what he intends to bring before you, and I take great pleasure, having known the doctor so long, and the methods of study which he pursues, in saying a few words in reference to his translation of this book. It contains information which you do not find in the principal American text-books. One of the subjects that the American text-books do not touch upon is the diagnosis of labyrinthine diseases and the functional tests. Dr. Holinger made a statement in the beginning of his talk that some one told him it takes too long to make a functional examination; it is not practical in this country, and I am the one who told him that. But the book which the doctor intends to translate will be of good to us all.

THE NOSE IN ITS RELATION TO EPILEPSY.

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Chance sent to me a year ago a patient suffering with epilepsy who had marked obstruction and pathological changes in the nose. This accident set me at working out what connection there might be between the nose and epileptic seizures.

I was fortunate enough to have access to a number of segregated cases of epileptic insanity at the Manhattan State Hospital. This group consisted of about 60 women. I made a careful rhinological examination, including the accessory sinuses, in 48 cases of this group. All were found to have extensive abnormalities of the nares of nearly every obstructive variety, and many had suppuration and sinuitis. The patients had been subjected to a great variety of treatment, both surgical and medical, in various institutions, extending over many years. Eighteen were selected from the number examined for observation and treatment of the nasal defects. The cases whose mental deterioration had not rendered them unresponsive and who had frequent epileptic seizures were chosen. The course of treatment was wholly operative, directed to the prevention of contact of intranasal structures. Five only of the 18 cases were observed; 2 of the 18 passed from observation. The remaining 11 were operated on from two to eight times each.

The rhinological work is not yet completed in any one case, but there is marked nasal improvement in all the patients operated upon thus far. The epileptic results are as yet very meager, but they show a slight decrease in the number of seizures, together with a slight improvement in the general mental and physical condition.

Case, No.	Age.	Years of epileptic seizures.	No. of months observed before commencement of treatment.	No. of months observed after commencement of treatment.	Average monthly seizures before treatment.	Average monthly seizures after treatment.	Decrease, Per Cent.
1	27	12	11	2	9	9	..
2	49	3+	10	3	4	3	25
3	22	12	11	2	25	22	12
4	33	6+	11	2	30	26	13
5	22	3+	9	4	52	17	67
6	38	30	3	8	38	19	50
7	36	28	5	3	14	10	28
8	24	5+	3	2	19	15	21
9	47	28	7	4	18	9	50
10	29	5+	9	3	25	17	32
11	46	8+	8	5	11	7	36
							Average, 30

I have observed at various times that blows upon the head and severe injuries to the upper spinal cord in man and animals often produce epileptiform convulsions. Also that a nasal cause of reflex epilepsy rarely exists in animals, among whom nasal obstruction and its consequences is practically unknown except in serious constitutional disease or some unusual parasitic nasal disease.

During the year I made the following conclusions, namely: that the number of seizures increased with increased congestion and obstruction of the nose and that they decreased when these symptoms diminished. I observed that colds in the head (nasal infection), lack of vasomotor tone and diminution of outdoor exercise were often associated with an increased number of epileptic convulsions. I also noticed that the fits did not occur when the Schneiderian membrane was anemic and hard, but that when they occurred the membrane was always congested and more or less distended. The engorged condition of the mucosa was noticeable before the attacks as well as after them. I further noted that in some patients painful manipulation of the nose at once produced seizures, which would recur in rapid succession if the irritation was repeated (3 cases). Cotton pledgets or crusts left in the nose increased the frequency of the attacks; nasal bleeding diminished the number of attacks. In a general way, whatever irritated the Schneiderian mucosa and made it congested and tumid was provocative of epileptic seizures; whatever tended to an anemia and contraction of this membrane tended to decrease the frequency of the seizures. The statistics of the Manhattan State Hospital show that epileptic seizures are slightly more frequent during the colder months.

From my observations I concluded that the stimulus for an epileptic seizure was apt to start in the nose. It seemed to me, too, that the epileptic seizures were reflex in character and that the epileptic reflex was the exaggeration of a normal nerve reaction under certain extreme stimulations.

Literature gives us only 52 cases of epilepsy definitely associated with nasal defects or disease. The treatment of these cases was not sufficiently long to justify more than the assertion that it had been a deterrent to attacks; very few have been observed long enough to justify the name of cure. If we look over the literature of the etiology of epilepsy we find that it is most common in the colder, densely populated countries, and least so in the warm, sparsely settled ones. I also gathered from the literature on epilepsy that overeating, indigestion, menstruation and mental excitement were exciting causes of increased frequency of seizures, and that these conditions have an important bearing on the circulatory condition of the nose.

A perusal of the literature showed that a small proportion of the reflex epilepsies had their peripheral origin in various parts of the body, while most of the cases noted were due to reflex irritation in or about the cranium, chiefly from the nose through the fifth cranial nerve, and that reflex epilepsies occurred occasionally in animals from various causes, among them nasal disease. In experimental epileptic seizures of cortical origin, the resultant nervous activity appeared to spread over a much larger part of the central nervous system than the area of direct stimulation, which suggested something of a reflex nature.

Severe injuries of the head and spinal cord have caused epilepsies in previously healthy individuals. Literature shows that animals may succumb to epilepsy when the stimulus is excessive; that they may be rendered more susceptible by weakening their resistance through previous experiment, and that they become unprotected against spontaneous epilepsy by experimental nervous lesions. The analogy holds good in man; a normal man becomes epileptic under severe stimulation, and a previous pathological condition, predisposition, or lack of protection is all that is necessary for slight exciting stimuli to produce epileptic seizures. Our conclusion from the literature is that the chief cause of reflex epilepsy in man is nasal irritation; that epilepsies due to the irritation of the cortex, the lower centers, or the cord, are, to a considerable extent, all reflex; that there is no essential difference between reflex epilepsy and essential epilepsy, or idiopathic epilepsy, except that the exciting cause is not obvious in the latter, and, finally, that epileptic seizures represent the exaggerated normal nerve reaction to certain stimuli.

The conclusions derived from my own observation and from the perusal of the literature are identical. A peculiar fact appears on consideration of the literature and after observation of the selected cases, namely, that epileptic seizures and the nasal condition fluctuate in unison. It appears both from my own experience and from the clinical and experimental literature that peripheral irritation of the fifth nerve—especially that portion supplying the nasal membrane—is more frequently the cause of epilepsy than the peripheral irritation of other nerves.

The nasal condition found in this series of cases of epileptic insanity—all long-standing cases of epilepsy, from 5 years to 30 years, all of whom had exhausted the known treatment of epilepsy—strengthens the author in his supposition that in these cases the nasal cause of peripheral epileptic irritation was the most resistant, exciting cause and at the same time the constantly acting one.

Reflex disturbances of the higher centers from irritation of the nasal branches of the fifth nerve are anamnesia, aprosexia, vertigo, migraine, hemicrania and scotoma scintillans, not to mention epilepsy; and of the lower centers, chorea, coughing, sneezing, temporary arrest of the heart beat, asthma and sexual reflexes. The great disturbances caused by comparatively slight nasal stimuli are worthy of note in this connection. The reason the fifth nerve, which supplies the Schneiderian membrane, is the most frequent conveyer of peripheral stimuli causing considerable central disturbance, including epilepsy, is, in the first place, because it is a cranial nerve of large size, with very extensive peripheral and reflex connections—practically the only sensory nerve supplying a region of the body which is, as a rule, pathological in civilized man; seconly, because the central connections of the fifth nerve bring it into peculiarly close relation with the higher and lower centers, and, thirdly, because it is the sensory nerve of a large part of the brain and meninges.

The weight of evidence favors the hypothesis that epilepsy is a reflex nervous symptom. It can be compared to vertigo, nausea or vomiting, which are caused by overstimulation of the peripheral space organs. These are normal nerve reactions to overstimulation of the peripheral space organ or of its center connection, but become pathological when excited by insufficient or trivial causes.

We explain the large number of cases having origin in intranasal irritation by the scientific hypothesis that, given a predisposition to epilepsy, the exciting peripheral irritation will probably first appear in the nose, not only because that organ is undergoing a retrograde change in civilized man—a condition rarely found in animals, but which when it does occur is fraught with the most dire consequences—but also because sensory stimulation of this organ has special effect on the central nervous system.

Epilepsy has always been a very resistant disease, or rather symptom, because its predisposing cause can rarely be removed, and its exciting cause, though occasionally found, is still less frequently removable. It has been overlooked where it most usually occurs and where it is most amenable to treatment, namely, in the nose, and here it has been most reprehensibly neglected.

If our view of epilepsy is correct, that it is a reflex symptom of central overstimulation, and that the cause rendering the patient abnormally susceptible is beyond reach, permanent cure may not follow the removal of the peripheral exciting irritant, because a disturbance in some other part may later again produce the exciting irritation.

The writer has essayed to treat this series of cases intranasally. He has obtained very slow results, which, however, have not been entirely discouraging since slight improvement has followed his efforts and since he has obtained additional proof of a peripheral cause of the seizures.

CONCLUSIONS.

1. Epilepsy is a normal symptom consequent on overstimulation of the higher centers of the cord and brain.

2. This overstimulation is largely reflex in its characteristics.

3. The normal nerve reaction may become pathological even when it is irritated by a comparatively insignificant peripheral stimulation.

4. Epileptic seizures are more often due to reflexes from the Schneiderian membrane through the fifth nerve than to other causes.

5. Appropriate treatment of the intranasal defects may be expected to lessen greatly the number of attacks, and in favorable cases to remove wholly the peripheral irritating cause of the epilepsy, which will allow cessation of the seizures.

6. In the epileptic neurosis, predisposition or weakened resistance of the central nervous system is due to other causes than the slight exciting peripheral irritations which have previously lessened the self-protective power of the higher nerve centers.

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A METHOD OF PREVENTING HEMORRHAGE DURING ADENECTOMY.

SAMUEL IGLAUER, B.S., M.D:

CINCINNATI, OHIO.

The operation for the removal of adenoid vegetations is usually attended by the loss of considerable volume of blood. If, as is usually the case, tonsillotomy be performed at the same time the total hemorrhage is of necessity increased.

Considering the fact that the adenoid patient is often a delicate anemic child, it would seem desirable to limit the operative hemorrhage if possible. There can be no doubt that a sudden loss of blood increases the danger of anesthetics[1] as well as the shock of an operation. Hemorrhage also increases the risk of infection, as proven by studies in experimental anemia in animals.[2]

One of the chief difficulties encountered by the operator during adenectomy and tonsillectomy is the hemorrhage obscuring the field of operation. In other branches of surgery the operators pride themselves upon minimizing the loss of blood, and there is no particular reason why the laryngologist should limit his endeavors in this direction. From a cosmetic point of view adenectomy is far removed from the domain of beautiful operations, and in our general hospitals now one seldom sees a considerable flow of blood except at the hands of the obstetrician and of the laryngologist.

Hitherto considerable attention has been paid to the position of the patient during adenectomy in order to avoid the aspiration of blood, but practically nothing has been done to prevent the hemorrhage itself.

TECHNIC.

In order to control the hemorrhage of adenectomy I have devised the following method:

It consists essentially in introducing the drawstrings of a postnasal tampon before beginning the operation and in drawing the tampon into place immediately following the adenectomy.

The instruments necessary for this procedure are, first, a modified Belloque canula made of a slender hard rubber Eustachian catheter. I have found the ordinary metal canula too thick and rigid for this purpose. Second, a rubber sponge of proper size to serve as a tampon. These sponges, owing to their elasticity, make excellent tampons and can readily be sterilized by boiling. A stout piece of tape is passed through the sponge, tied about it and left with the ends about a foot long. The patient is anesthetized until the pharyngeal reflex is partially abolished.

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[1] Hewitt: Anesthetics and Their Administration.

[2] Albutt Syst. Med. See Predisposition.

With the canula introduced through the nose, one end of the tape is withdrawn through the nasopharynx and out of the nostril. The other end of the tape with the tampon attached is allowed to hang out of the mouth. An assistant now makes traction upon the two ends of the tape, which thus serves as an admirable plate retractor, giving the operator more room and permitting of the use of the curette or a forceps with but little danger of catching the velum.

The adenectomy is now performed in the usual manner. Immediately thereafter the tampon is forced into place by the assistant drawing on the nasal end of the tape, while the surgeon with his index finger crowds the plug into the nasopharynx. Owing to the size and elasticity of the tampon the hemorrhage is immediately controlled. The operator can now proceed with the removal of the tonsils, if these are enlarged. In case the tonsils are very large they should be removed before beginning the adenoid operation.

The tampon is allowed to remain in position for a few minutes after the adenectomy, when it may be removed, as there is but little tendency to hemorrhage after the tampon pressure has been maintained for a short time. The tampon is easily withdrawn by pulling on the one end of the tape which has been left long for this purpose.

REPORTS OF CASES.

I have notes of eighteen cases in which I have employed this method. The patients ranged in age from 4 to 17 years. Gas and ether, or ethyl chlorid and ether, were employed in all of the cases except one case of ethyl chlorid anesthesia. In practically all the cases the tonsils were removed at the same time as the adenoids, and this may account for the blood vomited in some of the cases. This vomiting, however, is recorded in the first five cases when the technic was being perfected and is noted but once in the succeeding primary cases. The hemorrhage was easily controlled in all the cases in which the tampon was properly inserted. There were no secondary hemorrhages. The Belloque canula should be introduced with great care in order not to wound the nose or the adenoids. With the ethyl chlorid case the anesthesia was too transient for this method. One or two patients insufficiently anesthetized seized hold of the drawstrings. There were no ear complications in these cases and no sequelæ traceable to the tamponade.

It must be admitted that certain disadvantages attend the method of operating described above. In the first place, it requires a somewhat more profound anesthesia than ordinarily employed for the adenoid operation. Secondly, owing to the time occupied in introducing the tampon, it can not be employed with evanescent anesthetics, such as ethyl chlorid or nitrous oxid. Finally, it lightly complicates an otherwise rather simple operation and requires the aid of a good assistant.

The advantages which result from the employment of the tampon method may be enumerated as follows:

1. The drawstrings acting as a palate retractor protect the velum and permit of a more thorough use of the adenoid forceps than is ordinarily the case.

2. The hemorrhage is checked immediately. The loss of blood is thus reduced to a minimum, decreasing the immediate risk of syncope and the subsequent chances of infection.

3. The field of operation is left anemic for tonsillotomy

4. Ether anesthesia (or gas ether anesthesia) much safer than chloroform, may be employed, since the cerebral engorgement resulting from ether inhalation is counteracted by the tampon.

5. Postoperative nausea and vomiting due to swallowed blood are partially obviated.

6. The danger of aspirating blood into the trachea is diminished.

These considerations, it would seem, make this method worthy of further trial and extension, so that some day combined adenectomy and tonsillotomy may become an almost bloodless operation.

DISCUSSION.

Dr. Spohn:—This is a subject that interests all of us. Nothing so tests the resourcefulness of a physician as a severe hemorrhage. I have never seen very much hemorrhage from adenoid operations. The time to prevent hemorrhage is to begin three or four weeks before the operation. This can be done in the large majority of cases. In other words, the case should be prepared for the work in hand. Extreme hemorrhage occurs only in the regular bleeders. It is true there are a few cases on record that have died from adenoid operations; but this is the class of cases that should have some special preparation before the operation. When the tonsils and the adenoids are removed under one anesthetic, the tonsils should always be removed first. The severe hemorrhage comes from the tonsils, and not the adenoids. It seems to me the hemorrhage would stop by the time the operator had the tampon applied. This is my method of stopping hemorrhage of the nose, when it can be controlled with ordinary means. I believe calcium chlorid, administered in 15 to 20 gr. doses every hour for three or four hours before the operation, will save more blood than any tamponade.

Dr. McCaw:—The doctor's method of controlling hemorrhage in adenectomy is certainly ingenious and he deserves credit for reporting it. The method I use, and I presume others have used, is the application of ice cloths over the face, which produces a quick contraction of the blood vessels and controls the bleeding to a certain extent. But on the other hand is the amount of hemorrhage we usually get objectionable? I do not believe it is, for the reason that when we operate for adenoids there is usually a good deal of Eustachian tube congestion with impairment of hearing, and many of these cases immediately after the operation show great improvement in hearing. And again, the doctor operates, I think, a little differently from most of us in taking out the adenoids first. It is my impression that the majority of operators remove the tonsils first, and I believe that most of the hemorrhage is from the tonsillar stumps. I always use deep anesthesia. To operate under primary or local anesthesia allows gagging of the throat muscles, which increases hemorrhage. By using deep anesthesia and ice cloths the usual amount of bleeding following adenectomy is not great and does good instead of harm in most cases, especially where the hearing is impaired.

Dr. Goldstein:—It seems to me that our prime object in perfecting any form of technic should be simplified as much as possible and not complicated. Dr. Iglauer's unique and ingenious contrivance for controlling hemorrhage by this method of plug of rubber sponge, instead of cotton or gauze, is a very clean and neat procedure, and a useful thing to have on hand in case of hemorrhage, but for adenectomy operations, the simpler the operation and the simpler the technic, the more satisfactory the result.

Dr. Willis, Indianapolis:—As an adjunct to the armamentarium of the office, in case of necessity, on account of severe hemorrhage during adenectomy. I believe this device is excellent. Lately I have read, or had it suggested to me, that it takes children who have been operated upon for adenoid, at times, months to recuperate from the result of the operation, on account of the loss of large amounts of blood. Provided this could actually be proved by blood count, and hemoglobin test, this means, mentioned by Dr. Iglauer to lessen the hemorrhage in removal of adenoid, as it were, making this a bloodless, rather than an unusually bloody operation, would be exceedingly practical, in such cases as might suffer severely from the usual depletion. The routine method of using a plug, I am inclined to think, is not to be considered, as from what I have heard said by those persons on whom I have been obliged to use such a plug for excessive hemorrhage, I can not but feel that the presence of the plug and removal of the plug are worse punishment to the patient than the adenectomy itself. I have of late, in all surgical procedures about the nose and throat, prior to the time of operation, taken a drop of blood from the patient's finger to determine the coagulation point and hemoglobin percentage, and it has given me a feeling of security when at the time of operation I knew the coagulation time of the patient's blood to be six, seven or eight minutes, that I would not have if I operated without such information.

Dr. Kyle:—I want to remark that Dr. Willis had told me of that scheme of his, and in a case I had, I took a drop of blood from the patient to ascertain the coagulation point, but while waiting for it to coagulate I went on with the operation, and before the blood had time to coagulate, the operation was over and there was practically no bleeding.

Dr. Holinger:—Did you ever have any infection of the middle ear from this tamponade? These children usually have trouble in their Eustachian tubes and middle-ears, which greatly favor acute suppuration.

Dr. Stucky:—A few years ago at the Mississippi Valley Medical Association meeting in Memphis, I made the statement that adenectomy and tonsillectomy are the most underestimated and underpaid operations in surgery, and I objected to their being classified as minor surgical operations. In twenty-six years of practice I have never seen serious hemorrhage from tonsillectomy but once. I never saw serious hemorrhage from adenectomy but once. Three weeks ago I removed a large soft, mushy adenoid mass from a young man, 18 years of age. I used nothing but the curette. It came out as easy as it would have come out of a child 4 years old. There was practically no fibrous tissue in it, and while it bled quite freely at the time it soon ceased, and he came from under the influence of the anesthetic rapidly. In eight hours they telephoned me from the hospital that he was spitting blood. Now, I had not touched the tonsils. I went out to see him, and was surprised to find him nearly exsanguinated; the blood was oozing down the posterior wall, and the whole field was deluged with blood. He was weak from the severe hemorrhage and could not be lifted from his pillow without fainting. I had to use the post-nasal tampon, but not like Dr. Iglauer's. I have never tried the rubber sponge. But that is the first time in twenty-six years that I have ever seen a hemorrhage that amounted to anything from a simple adenectomy. I think when we consider the advantages derived from this operation, the good that an adenectomy or tonsillectomy does for the present and future of the child, especially the health and the hearing, and everything connected with the life and happiness of the child, I think we, as medical men, should cease to classify adenectomy and tonsillectomy as minor surgical operations. This patient I speak of was not a bleeder, the hemorrhage was venous, and of such an alarming nature as to require tamponade, and a tedious recovery was the result.

Dr. Iglauer (closing):—At this late hour I did not expect very much discussion of my paper. If you will sum up what the gentlemen have said, it seems to me they have all made a plea for hemorrhage. They all say, what is the use of stopping the hemorrhage? I reply, what is the advantage of having the hemorrhage? Now, if it were a complicated method that I advocate, the contention might be correct, that it isn't worth while. However, it is a simple thing to pull a draw string out of the nose and to insert a tampon. As to the quantity of blood lost during adenectomy, I think there is considerable, considering the weight of the child; especially if one employs ether anesthesia, which is safer than anything else. If chloroform is used the hemorrhage increases the danger of chloroform syncope. I have seen children vomit large quantities of blood after this operation, and there is no advantage in this hemorrhage. We know hemorrhage increases the risk of infection, and perhaps lowers the opsonic index. I do not believe that ice cloths will check a profuse hemorrhage. They may gradually stop it, but not at once. The surgeon stops it with a hemostat, and if one can check the hemorrhage with mechanical pressure, why not do so? If one operates in a private house, it must be admitted that the house, or the room rather, looks rather bloody after the operation. Dr. McCaw objects to removing the adenoids before the tonsils. If the tonsils are very large I remove them first, otherwise I do not find it necessary to do so. The tonsillar hemorrhage is more easily controlled by position than the adenoid hemorrhage. The adenoid blood is more apt to run into the larynx, but the tonsillar blood flows into the mouth. I think this method should include control of the tonsillar hemorrhage if possible. One gentlemen spoke of the tendency towards complicated surgery and that we should progress toward simplified surgery. I think the more surgery advances the more complicated are the operations and the more complicated the technic. In oral surgery we formerly employed simple instruments and simple methods, but now we employ more and more complicated apparatus. The suggestion of giving chlorid of calcium as a prophylactic seems a very good one, but as a rule our adenoid cases do not receive much preparation. The diagnosis is usually made one day and the operation the next, or as soon thereafter as possible. The coagulation test in some cases would detect a bleeder and might prevent using a plug, or anything else. One gentleman objected to the plug owing to its discomfort. He forgets that these patients are under general anesthesia, and before they come out from the anesthesia the plug is removed, so that objection does not hold. Dr. Holinger inquired about the ear. I stated in the paper there were no complications in the ear. It occurred to me the presence of a plug might drive some blood up into the ear, but such a thing has not occurred. It was also suggested to use nitrate of silver as a styptic. I think it inadvisable to use an escharotic, because infection is more apt to occur with the use of these chemicals. The rubber tampon, aside from its use during adenectomy, will be found advantageous in intranasal work under general anesthesia, as it effectually prevents any blood from escaping into the pharynx.

EXHIBITION OF INSTRUMENTS.

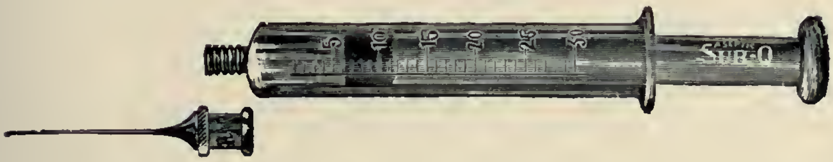
NEW NEEDLES FOR PAINLESS, BLOODLESS TONSIL DISSECTION IN ADULTS.

DERRICK T. VAIL, M.D.

CINCINNATI.

I have found these needles of my own design of great use in rendering tonsil, subtonsillar and peritonsillar tissues anesthetic and anemic preparatory for intracapsular dissection in adolescents and adults.

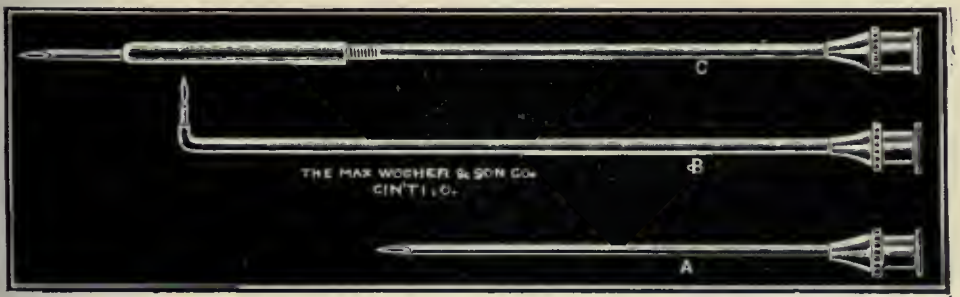
I use the Sub-Q all glass syringe, which has asbestos packing, sold by the Max Wocher & Sons Co., of Cincinnati. It is boiled and so are the needles just prior to the operation.



The solution used is as follows:

℞. Cocain muriat, 2 per cent. solution....................ʒi
Adnephrin, 1/2,000 (Stearns) ....ʒi

This solution is also boiled just prior to the operation and is prepared fresh each time. It will be noticed that the solution reduces the cocain to 1 per cent. and the adnephrin to 1/4000.



With the guarded straight needle "C," about twenty drops are injected in and around the tonsil through the anterior pillar; much of the solution escapes through the tonsil crypts.

With the angular needle "B" about ten drops are injected carefully under the basement capsule of the tonsil, which is reached through the bottom crypts, through the base of the supra-tonsillar fossa and between the anterior pillar and the body of the tonsil. The effect is that the tonsil is instantly blanched and rendered completely anesthetic, and the dissection, which is accomplished with an exceedingly sharp scalpel or pointed bistoury, renders this a most satisfactory operation. The upper one-half or two-thirds of the tonsil is turned out of its bed in this way and the Peters snare or Ballenger écraseur severs the base, which is like a pedicle.

Owing to the presence of the large vessels near the lower part of the tonsil, sharp cutting instruments should be avoided in severing the pedicle.

FORCEPS, HAND BURR AND PERITONSILLAR ABSCESS PERFORATOR.

J. A. Stucky, M.D.
LEXINGTON, KY.

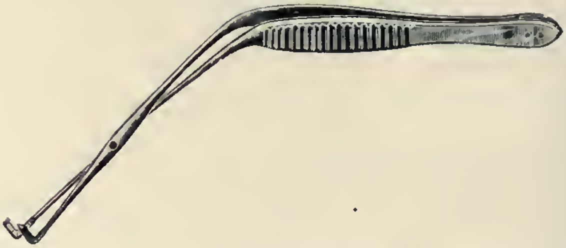


Fig. 1.—Forceps for removing ossicles in doing the Stacke operation.

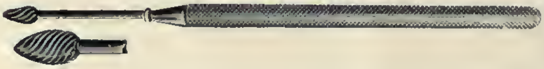


Fig. 2.—Hand burr, olive pointed, for rimming out the tympanic orifice of the Eustachian tube.

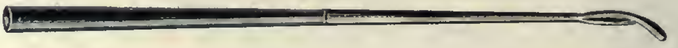


Fig. 3.—Peritonsillar abscess perforator.

TRANSACTIONS

of the

TWELFTH ANNUAL MEETING

of the

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGICAL DIVISION

HELD AT
LOUISVILLE, KY.
SEPTEMBER 26, 27, 28, 1907.

OCULAR SYMPTOMS OF INTRACRANIAL COMPLICATIONS IN OTITIC DISEASE.

PERCY FRIDENBERG, M.D.
NEW YORK.

The progress of otology during the past ten years has been so largely practical and surgical that even the operations for intracranial complications have lost much of their originally formidable, not to say ominous, aspect. The otologist of to-day is, theoretically and technically, prepared, in case of definite indications, to follow up the suppuration from its source in the middle ear or mastoid whithersoever it may lead him. It is now generally recognized that complicated methods of localization and the use of base lines, measurements and cranial topographical studies are of minor importance, as we have learned that the intracranial pus accumulations are, in a majority of cases, to be found in the immediate neighborhood of the purulent infectious focus in the temporal bone, or at least that they can most easily be reached, treated and drained by an extension of the original field of operation. While this increase of security in operating and the consequent improvement in surgical statistics render the question of diagnosis less immediately vital and important, they by no means lessen its scientific import or make it any less incumbent to place upon a definite basis, as far as possible, the question of the significance of intraocular changes in the diagnosis and prognosis of endocardial complications of purulent otitis.

An interesting feature in the study of ocular symptoms in otitic intracranial complications and one of importance in the history of investigations on this subject is the fact that in this country many ophthalmologists are aural surgeons as well. A generation ago, when the surgery of otitic disease was in its infancy, this was almost invariably the case, and it is due in large measure to the work of Kipp, Gruening, Knapp, Roosa, Alt and other Americans that the teachings of Politzer, Bezold, Zaufal, Lucae, Troeltsch and Schwartze have been spread in this country. Kipp was, in fact, the very first to call attention to the importance of making ophthalmoscopic examinations in cases of ear disease. His first article[1] on this subject antedated that of Zaufal by a year, yet the latter is always spoken of, at least by German authors, as the pioneer in this field. Kipp's second article[2] appeared in 1885, and the conclusions as to the value of ophthalmoscopic changes in prognosis and treatment stated in his second paper are now generally accepted. Hansen urges the importance of the otologist being skilled in ophthalmoscopy in order to insure early and repeated examination of each and every case. American otology recognized and fulfilled this requirement years before it was formulated by Hansen in Germany.

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[1] Archives of Otology, vol. viii, 1879, p. 147.

Diagnostic signs are of practical import, whatever their scientific and theoretic value may be, as far as they serve as a basis for therapeutic considerations, prognosis or operation. This does not imply that a study of symptoms in cases of diseases which for other reasons are beyond the reach of therapeutics, at the time, is lacking in academic interest or in eventual practical importance, as the progress of methods of treatment may finally come abreast of the diagnostic methods. In the sphere of brain surgery or that part of it associated with the intracranial complications of otitic disease, the practical indications to explore the cranial cavity and determine the condition of sinuses, meninges and central organs are quite definite and generally urgent. The making of an accurate diagnosis hardly aids in treatment in this field. On the other hand, there is a development of other diagnostic methods, among which we may mention lumbar puncture, the blood examination and differential leucocyte count, the careful examination of the reflexes, Kernig's and Babinski's signs, the careful bacteriologic examination of aural discharge and of tissues removed from mastoid, sinus or jugular vein at the time of operation.

Inflammatory changes or circulatory disturbances in the optic nerve have always been considered corroborative evidence of similar endocranial conditions causing an increase of brain pressure or indicating inflammatory processes. In the otitic complications the evidence is often contradictory, obscure or insufficient. The views of the most experienced observers and the opinions expressed by standard authors on this point do not agree perfectly.

Bacon.[3]—Temporo sphenoidal abscess. Optical aphasia a symptom of the highest diagnostic value. The patient, when shown an object, knows it, but is unable to name it (better, has no conception of its name; the other phrase might mean that he could not speak the name he had in mind, i. e., motor aphasia), but he is able to recognize and understand its name, and will often describe the object. He can sometimes name the object if allowed to handle or smell it (showing there is no motor aphasia, and that a conception of name, due to stimulation of memory pictures, can be accomplished over other paths than the visual). In one case there was, besides the difficulty in naming an object, difficulty in calling to mind its appearance, visual amnesia (Starr), "due to a suspension of function in association tracts lying deep in white matter of temporo-occipital lobes."

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[2] Ibid., vol. xiv, 1885, p. 1.

[3] Diseases of Ear, 1902.

Inflammation of optic nerve may or may not be present. More frequent in cerebellar abscess. Symptoms of latter may be simulated by disease of semicircular canals. In the case of a child with suspected cerebellar abscess characteristic symptoms, including choked disc with retinitis, were entirely abolished by removal of granulations from the middle ears and establishment of proper drainage. In an abscess of the right cerebellar hemisphere there was edema of the right optic nerve. Photophobia and nystagmus may occur.

Sinus Thrombosis.—When T. has extended to cavernous sinus and ophthalmic vein has become engorged, there is likely to be a certain amount of edema of the eyelids on the corresponding side. Neuro-retinitis is noticed in some cases, more especially in the later stages, but it is not constant.

Purulent leptomeningitis. Photophobia, pupils contracted and responding but little to light, and optic neuritis. Later the pupils are dilated and often unequal. In children, symptoms suggestive of meningitis may be due to retention of pus in the middle ear. When paralyses occur, and there are optic neuritis and other characteristic symptoms, the diagnosis is assured. (Koerner does not lay much stress on optic neuritis in meningeal irritation in children, and cites a case in which he was urged to operate on account of double choked disc. It was a serous meningitis of mild type, and, as pulse and G. C. were good and weight was increasing, he temporized. Recovery was spontaneous.)

Blau.[4]—It is advisable to consider any disturbance of ocular motility occurring in the course of an otitis media as suggestive evidence, symptom of secondary involvement of the labyrinth or of intracranial complications. For the diagnostic use (Verwerthung) of ocular symptoms in the diagnosis of intracranial complications, it is of prime importance to determine whether morbid changes, particularly of the fundus, can be induced by middle-ear disease. Cases cited to substantiate such a possibility were all suspicious of already existing complications, and were so considered by the observers themselves. Ocular symptoms are, then, of definite significance, to the extent that they appear to develop only when otitis has involved the interior of the skull, with the exception of nystagmus, which may be excited as a reflex on irritation of the "tonus labyrinth" (semicircular canals). Hyperemia of the nerve, neuritis, choked disc, render it highly probable that we have to deal with threatening or existing intracranial complications, but do not justify any conclusions as to their nature. On the other hand, the absence of intraocular changes by no means excludes the presence of intracranial suppuration, so that in the presence of other characteristic symptoms the negative result of ophthalmoscope has absolutely no significance. It is of importance as corroborating the other symptoms and clinching the diagnosis when present.

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[4] Encyclopedie d. Ohrenh. Art.. Auge. Ostmann, 1900.

Burnett[5] quotes T. R. Pooley to the effect that "if optic neuritis is found in connection with purulent inflammation of the ear the diagnosis of extension of ear disease to the brain is certain, no matter whether other evidence exists or not." Unfortunately it does not explain the nature of the intracranial lesion, and, while optic neuritis in chronic otorrhea, or even acute otitis, would be an urgent indication for immediate mastoid opening, its occurrence must be considered only in connection with other symptoms as an indication for an exploratory opening into the cranium.

Optic neuritis is usually not observed, or only slightly marked, in uncomplicated sinus thrombosis; it is sometimes found with extradural abscess. In cavernous sinus thrombosis, due to the passage of infection from the middle ear by the carotid canal (Koerner) edema about brow and orbit, exophthalmos, paralysis of extraocular muscles, immobility of the globe (due to orbital cellulitis), edema of lids, chemosis of conjunctiva and choked disc on the side of the chronic aural suppuration, are seen.

In otitic cerebral abscess and in extradural abscess neuritis is frequent. Optic acoustic aphasia is usual in abscess of left temporal lobe, or in the right lobe if the patient be left handed. Ptosis of left upper lid has been seen in left temporal lobe abscess (Steinbruegge). Sensory aphasia with homonymous hemianopsia with preservation of pupillary reaction points to temporal lobe.

Symptoms of extradural abscess or of sinus thrombosis always dominate and mask those of brain abscess. This applies to fundus changes also.

Cerebellar abscess shows varying pupils, the wider being on the side of the abscess, conjugate deviation away from affected side, horizontal nystagmus, abducens paralysis, lagophthalmus, due to facial paralysis. Double optic neuritis is often the only symptom, but the fundus may remain normal even in fatal cases.[6] Cerebellar abscess is very likely to obstruct circulation of cavernous sinus and ophthalmic vein.

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[5] Diseases of Ear, 1901.
[6] Woodward, Arch. f. Otol., January, 1906.

Meningitis. No mention of ocular symptoms.

Bezold.[7]—Labyrinthine suppuration shows, at least in early stages, characteristic symptoms of irritation, subjective dizziness, vertigo even while lying down, disturbance of gait, horizontal nystagmus when gaze is directed to the sound side, and vomiting. Nystagmus may be rotary and always disappears (destruction of ampullar organs) sooner than the dizziness. Often complicated by fatal meningitis and cerebellar abscess.

Extradural pus collections may give no clinical signs.

Operate after eight weeks in acute otitis, and after several months of fruitless treatment of fetid discharge in chronic cases to find the source of continued suppuration.

Choked discs and double edema of both lids speak for thrombosis of both cavernous sinuses.

In brain abscess, optic neuritis, oculomotor or abducens paralysis. Otitis leptomeningitis may start from any point of a sinus thrombosis, or from brain abscess which has ruptured into ventricle or subarachnoid space. Extradural abscess may cause necrosis of dura and spread to arachnoid and pia.

Dench. Serous Meningitis (1903).—Most frequent paralyses those of the third, fourth and sixth nerves. Paralysis of the third nerve causes at first contraction, and later dilatation of the pupil. An early symptom is failure to respond to light. Where local paresis exists, divergent strabismus is probably the most frequent symptom. The fundus will most generally show either a choked disc or an incipient swelling of the optic papilla. It is strong corroboration of the other symptoms of meningitis, but is found in any intracranial lesion which increases pressure. Sinus thrombosis. Presence of choked disc will confirm the diagnosis in doubtful cases.

Brain abscess. Choked disc may be present, but is not characteristic of abscess alone.

Cerebellar abscess. Choked disc is often present.

Brieger.[8]—Fundus changes generally missing in uncomplicated extradural abscess, and arouse suspicion of other endocranial complications. Extradural compression causes disturbances in fundus which are rapidly compensated. Repeated examination may detect transitory fundus changes. Abducens paralysis is not infrequent.

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[7] Lehrb. d. Ohrenh., 1906.

[8] Otogene Erkrank. d. Hirn., 1903.

Kessel reports paralysis of abducens and oculomotor nerve of the opposite side, due to collateral edema of meninges or brain, and not to direct compression.

Purulent leptomeningitis. While Pitt found the fundus normal in all uncomplicated cases, while Knies claims optic neuritis as the most valuable diagnostic aid, the truth lies in the middle. His experience agrees with that of Hansen, that about half the cases show changes of the fundus which are sufficiently marked to allow a diagnosis. But these changes never appear as typical choked disc. They are rather in the nature of optic neuritis, and from slight hyperemia to entire obscuration of the margin of the disc. Generally double, occasionally unilateral, or unequal in degree, or even more marked on the side of the sound ear. In the cases with insidious onset optic nerve changes were rarely seen at an early stage before manifest symptoms developed. The pupils show a markedly rapid variation between contraction and maximum dilatation.

Abducens and oculomotor paralysis are not always due to localization of the purulent process at the base, but may be due, in still circumscribed meningitis, to general increase of pressure in the cerebrospinal fluid and be present as the only symptom for some time before the meningitis becomes generalized.

Nystagmus may be present in the form characteristic of labyrinthine disease or, more correctly, of implication of the semicircular canals, i. e., horizontal oscillations when the gaze is directed toward the sound side. After generalization of the purulent process, and not infrequently after operation, the nystagmus also becomes generalized in the sense of occurring with all ocular rotations, either constantly, and even when the eyes are at rest or the lids closed, or only when an object is fixed. This would indicate a spreading of the suppuration from the horizontal to the other two semicircular canals, independent of movements of the eye.

Conjugate deviation is rare.

Serous meningitis. Eye symptoms, as neuritis of slight degree, never as choked disc, with only slight hyperemia of the papilla. May be transitory or develop early, but may also appear later (Hegen) even several days after increase of intracranial pressure had been relieved by operation. Abducens paralysis may be the only symptom of local serous meningitis, due to compression at the base. A case of nuclear lesion on the floor of the fourth ventricle, causing paralysis of associated motion (Blick-laehmung) has been reported by Brieger.

Kirchner.[9]—Cavernous sinus thrombosis may develop early not only in chronic but in acute otitis media, especially after influenza. Exophthalmos, edema of the lids, paralysis of extraocular muscles, disturbance of vision, are the usual symptoms.

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[9] Ohrenhk., 1904.

Kyle, J. J.[10]—Purulent leptomeningitis. Contraction of pupils, loss of light reflex, later dilatation.

Thrombosis. Optic neuritis is one of the symptoms (quoted from Ballance). Extradural abscess. Occasionally optic neuritis a suggestive symptom.

Love, J. K.[11]—Meningitis. Pupils contracted, reaction to light, lessened. Later, optic neuritis is often present, pupils unequal and dilated. Ptosis and squinting may occur.

Sinus thrombosis may simulate meningitis. Optic neuritis may be present. *Brain abscess.* In early, irritation stage, pupils contracted, photophobia. Later unequal and do not respond to light. In tempero-sphenoidal abscess, third nerve may be involved, and ptosis, accommodation, paralysis and oculomotor paralysis of all but external rectus (abducens) occur. Optic neuritis occurs in about half the cases of cerebral and 60 per cent. of cerebellar abscess.

Koerner.[12]—*Extradural abscess.* Neuritis not unusual (Pitt, Lane, Johnson, Zaufal). Of 11 uncomplicated cases, 8 normal. Of 20 uncomplicated in Schwartze's clinic (Grunert), all normal. Koerner cites two cases, however, of marked changes, both in children, with extradural abscess in posterior fossa. In one case the neuritis increased after evacuation of the abscess, particularly on the ear-sound side. In the other, double choked disc, also more marked on the sound side, first appeared after operation. Cure in both. There was a possible complication with sinus affection in both.

Knies' view as to the importance of optic neuritis in diagnosis of meningitis is not shared by Koerner. The cases of Kipp and of Andrews had no postmortem, and the histories read like complication with sinus phlebitis and extradural abscess, respectively. In serous meningitis, particularly in children, optic neuritis has been observed in light as well as in severe cases. This is the more surprising, as optic neuritis is missed so often in purulent meningitis.

Thrombosis. Optic neuritis and choked disc common in cavernous sinus thrombosis. K. has not seen it in sigmoid sinus thrombosis uncomplicated. Leutert saw O. N. in 1 of 10 cases. Hansen, 3 in 8 uncomplicated cases. Jansen finds the changes often and finds them of diagnostic value.

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[10] Diseases of the Ear, etc.1906.

[11] Diseases of the Ear, 1905.

[12] Otit. Erkrank. d. Hirns, etc., 1902.

Jansen has seen nystagmus in sinus thrombosis and extradural abscess.

Brain Abscess. Neuritis frequent, but not invariable. Generally double and more marked on the side of the abscess. Late symptom and not marked, and may advance after evacuation of the abscess. Sight often but slightly affected, and consecutive atrophy rare. Hansen, 12 cerebral abscess, 2 with vessel changes and 4 with O. N.; 7 cerebellar, 2 O. N. In one of the latter, neuritis only on side of abscess, in 3 double and equal, in 4 more marked on abscess side.

Total amaurosis seen in 3 cases by MacEwen and Berridge (reported in detail by Koch, Otit. Kleinhirn Abscess., Berl., 1897). Operation in one case, with disappearance of amaurosis. In one fatal case there was complicating ventricular serous meningitis and purulent leptomeningitis. Case 3, P. M. incomplete. Nothing similar has been observed in cerebral abscess.

Hemianopsia generally due to lesion of the internal capsule, or when situated in occipital lobe to subcortical lesion of the optic radiation. These are distant symptoms due to pressure of abscess on neighboring regions and edema of brain substance.

Oculomotor palsy frequent. Temporo-sphenoidal abscess of at all considerable size invariably causes paralysis of some side, generally incomplete and limited to pupillary fibers and levator (ptosis and accommodation paralysis), occasionally involving all branches.

Pupillary mydriasis common in cerebellar abscess.

Abducens paralysis may be homolateral or crossed. It is of little diagnostic value, occurring in serous meningitis and spontaneously curing complications.

Conjugate deviation may be toward side of abscess (Kalmus, Moos) or away from it (Schwartze, Acland and Ballance, Hansberg, Winter and Deansley). Nystagmus (Mueller, Winter and Deansley, Ballance, Barling, Okada). It is a question whether this is not a labyrinthine symptom.

Partial or complete oculomotor paralysis is a valuable sign of temporo-sphenoidal abscess.

In children brain tumor (tubercle) not infrequently complicates suppuration in the temporal bone. Choked disc speaks more for tumor, neuritis for abscess. Chorioidal tubercle may be discovered.

Knapp, H.[13]—Pachymeningitis externa; epidural or extradural abscess. Symptoms of meningitis are present in irritation in many cases—headache, sl. rise in T., choked optic disc, pressure pain, somnolence, slowing of pulse, stiffness of neck, but these symptoms are too indefinite to make a diagnosis.

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[13] De Schweinitz and Randall, System. Art. Complications of Tympanic Inflammation, 1899.

Pachymeningitis interna, subdural or intradural abscess with softening and abscess of adjacent brain substance—circumscribed encephalitis—occurs when pia and arachnoid are agglutinated over inner surface of an infected area of the dura and exudation or perforation takes place. If not adherent purulent leptomeningitis results. Optic neuritis is rare.

Sinus thrombosis and pyemia. As the last of eight symptoms in importance and frequency, K. mentions stupor and optic neuritis as present only in complication with encephalic abscess and meningitis.

Brain abscess, four stages, initial, manifest, latent, terminal. Optic neuritis, earlier and more pronounced on the diseased side, is one of the symptoms of brain pressure.

Homonymous hemianopsia, on record seven times, probably would have been found oftener if looked for regularly.

Partial or total paralysis of the motor communis oculi and abducens nerves, conjugated deviation of the eyes (to either side), and nystagmus, are rare and indefinite symptoms.

Lake, R.[14]—Meningitis. In progressive cases, intolerance of light, at times amounting to photophobia, becomes marked. The pupils, which are commonly unequal, react to light, but usually more or less slowly than normal. Optic neuritis or papillitis will in most cases be noted. After development of the lethargic state the pupils often become widely though unequally dilated, and all reaction to light is gradually lost, while in basic cases a tendency to squinting is more frequently noted. Nystagmus may be present in later stages.

Extradural abscess. It is rare to find optic papillitis in uncomplicated cases.

Brain abscess. Pupil on affected side frequently contracted and only reacts sluggishly to light and accommodation, in which case the abscess will be found to be small, relatively, but if it is of large size the pupil on the same side will be both dilated and stable. Paralysis of the third nerve on the same side as the abscess is a variable sign; optic neuritis, or papillitis, is found during the whole of this stage and may be only on the affected side.

Cerebellar abscess. In second stage there is not infrequently conjugate deviation of the eyes to the side opposite to the lesion, along with optic neuritis. Lateral nystagmus with large excursions is especially frequent in lateral lobe abscess, the movements of the eyeball being toward the side on which the abscess is situated. Rotary nystagmus points toward involvement of the middle lobe. (These considerations seem to be largely hypothetical. If the movements of the eyeball are toward the side, any side, there must be deviation, if not, there is merely horizontal nystagmus. L. may mean that nystagmus develops only when the eye looks toward the affected side.—P. H. F.).

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[14] Diseases of the Ear, 1903.

A final decision on such points is often impossible until a large number of cases has been collected, critically studied, classified and analyzed and a statistical study made of the results. Statistical studies of this sort have been made in Germany by Sessous, Hansen, and by Koerner, whose cases were supplemented a year or two later by Takabatake. In this country there have been no analyses of large series of cases, although various otologists have reported their observations of specially interesting, grave or successfully treated cases of brain abscess, meningitis and sinus thrombosis.

Hansen's statistics comprise 97 cases, of which 45, about 46.4 per cent. showed optic nerve changes, while Koerner reports 54 cases with positive ophthalmoscopic findings in 16, or 29.6 per cent. Sessous (Lucae Festschrift, 1905) discusses 104 cases, 5 per cent. of all those treated at the Berlin clinic from 1895 to 1905. Ophthalmoscopic changes were most frequently noted in meningitis complicated with sinus thrombosis, least often in extradural abscess. A normal fundus is no proof of absence of intracranial complication, nor does the presence of fundus changes allow us to draw any but the most limited and cautious conclusions as to their presence. This also holds true in regard to prognosis as to recovery. Fundus changes are of diagnostic importance only when other cerebral symptoms are lacking. This is in marked contrast to the view of Ostmann cited above, that fundus changes clinch a diagnosis already made probable by other symptoms, but are of no value by themselves, and to the opinion of Koerner and of Hansen, who attach great importance to positive findings in the ocular fundus in suspected cases of intracranial suppuration. Hansen sums up his observations as follows: "Fundus changes may occur in any intracranial complication. Least frequently (18 per cent.) in extradural abscess, and then more often in the deep seated than in the perisinuous variety, they are most often observed, 87.5 per cent., in cases which had been considered as hyperemia of the brain and meninges, or serous meningitis, in 57 per cent. of uncomplicated, and 47 per cent. of complicated purulent meningitis (about 50 per cent. of all meningitis), in 50 per cent. of cerebral, and 29 per cent. of cerebellar abscess, 37.5 per cent. of uncomplicated and 47.5 per cent. of complicated sinus thrombosis (agreeing with Jansen). In other words, fundus changes are about equally frequent in the various forms of otogenous intracranial suppuration, occurring in about 44 per cent. Neuritis is the most common condition, slight neuritic change, vessel changes on the disc next in frequency, while real choked disc is unusually rare (3 per cent.) and occurs only under certain definite conditions. Both eyes were affected in the great majority (80 per cent.) of positive cases, and the changes were usually (75 per cent.) more marked on the affected side, although the opposite condition was noted in 9 per cent. Unilateral involvement of the fundus, observed in 20 per cent. of the positive cases, was invariably situated on the side of the ear affection. In brain abscess the fundus changes were almost invariably (87.5 per cent.) bilateral, and more marked on the affected side (50 per cent.) or of equal intensity (37.5 per cent.). Hansen does not agree with Koerner, MacEwen and Oppenheim that fundus changes in brain abscess are not infrequently found on the side opposite the intracranial lesion. As other symptoms of intracranial complications are often variable, evanescent, scanty and obscure, and the recognition of these ominous conditions consequently difficult and uncertain, positive evidence of fundus changes, which can always be detected when present (except in cases of opacity of the media, as cataract, etc.) is of undeniable and decided value. Their development in otitis suppuration always indicates the presence of some intracranial complication, for it is an unproven and improbable assumption that middle-ear disease alone, without endocranial suppuration, can induce morbid changes in the optic disc. As such changes are found in only about one-half of all cases of intracranial complication, their absence is never to be considered as positive evidence. Fundus changes are of definite importance in suppurative otitis, as their development, even in the absence of all other cerebral symptoms, indicates some intracranial complication, and accordingly a more severe affection. The prognosis of intracranial complication itself, on the other hand, is not influenced by the presence of fundus changes, as the latter may occur in the least dangerous as well in the most serious forms, and in mild as well as in severe or even fatal cases of a single form. Ophthalmoscopic examination should be made in every case of otitis suppuration, and repeated frequently in case there is any unusual feature in the progress of the case or even the slightest cerebral disturbance. The otologist should carry out these examinations himself, as this is the only guarantee for their regular methodical performance and record. Disc changes are an immediate indication for opening the mastoid in a case of middle-ear suppuration which for any reason has not been operated. The subsequent course of the case must then decide whether further surgical intervention is necessary.

Koerner agrees in the main with Hansen as to fundus changes being bilateral, even when the intracranial disease is limited to one side, in the great majority of cases, and as to the presence or absence of fundus changes having no significance for the prognosis of the intracranial complication. In purulent meningitis he comes to quite different conclusions, all of his 6 uncomplicated cases having had normal optic discs up to the time of death, while of the 8 complicated cases 6 showed fundus changes. Simple thrombosis caused disc changes in only 1 of 5 cases. One-sided occlusion of the cranial sinuses, it seems, is not sufficient to produce venous congestion in the eye ground, and this applies as well to the sudden disturbance of circulation incident to jugular ligation as to the more gradual stoppage by thrombosis or endophlebitis. Only about 25 per cent. of all cases of intracranial suppuration showed optic nerve changes. They are more generally absent than present, and more apt to indicate a combination of conditions than any one complication. Thus of 18 cases of a single complication only 3 showed fundus changes, and even in these there was some suspicion of another form of intracranial suppuration. Koerner sums up as follows: The variety of fundus change, neuritis and choked disc, respectively, does not allow any conclusions as to the character of the intracranial complication. A more marked development of fundus changes on one side does not indicate exclusive or even predominant involvement of the corresponding half of the cranium. Fundus changes do not influence the prognosis, and even advance of these changes after evacuation of pus in the cranial cavity does not alone make the outlook any worse.

My statistics and conclusions are based on a study of the records of the New York Eye and Ear Infirmary from 1905 to 1907. Of a total of 19,129 cases of ear disease treated during these two years 11,176 were affections of the middle ear and Eustachian tube, 1,064 affections of the mastoid. There were 11 cases of cerebral, 25 of epidural, and 73 of subperiosteal abscess; 12 of jugular vein and 13 of lateral sinus thrombosis. These figures represent the cases so diagnosed on admission. The full number of complicating intracranial cases is to be determined only from the report of operations which is shown on Table 1, which shows 16 exploratory and 65 curative operations for sinus thrombosis, 24 exploratory and

6 curative for brain abscess; 31 ligations of the internal, and 1 of the external, jugular, and 2 extirpations of the internal ear for labyrinthine suppuration. These cases were operated on by or in the service of Drs. Dench, McKernon, Bacon, Whiting, Lewis, Adams, W. S. Bryant, Rae, J. D. Richards, Stevens, Kenefick and Brandegee.[15]

TABLE 1.

Diseases.	Death.		Recovery.	
	Fundus Changes.	Fundus Normal.	Fundus Changes.	Fundus Normal.
I.—Extra-dural abscess.	......	......	......	2
II.—Purulent leptomeningitis	......	......	......	4
(a) Uncomplicated	4.1 †(1)	1		
(b) Complicated with	†1) (1)			
1. Brain abscess.	(1)	1	(1)	
2. Cerebral abscess	((1)	°1 ?1?		
3. Sinus thrombo is	2 (1) ((1)			
4. Epidural abscess (R. and L.).	(1)			
III.—Sinus thrombosis.				
(a) Uncomplicated...	6, ‡ (1)	......	6.1) (2)	
(b) Complicated with				
1. Perisinus abscess.	((1)	1	(1) ?1)	5
2. Extradoral abscess, middle fossa	......	......		
3. Sinus thrombosis (general) and meningitis.	1)			
4. Cavernous sinus	(1†			
IV.—Brain abscess.				
Cerebral.	1	1		
Cerebellar.	......	3		
V.—Cerebral disturbance in otitis without intracranial complications.	......	......	1 (1*)	1 1?
Erysipeles.	......	......	(1)	2
Abscess of temporal muscle.	......	4		

Explanation of abbreviations and signs.—(,), ()=neuritis; †=choked disc; ‡=thrombosis of central retinal vein and ophthalmic vein; °=ophthalmoscopic examination impossible (media opaque, etc.); *=double atrophy, post-neuritic. Doubling of signs, as (( )) ‡‡ indicates marked fundus changes. A sign before the number indicates fundus changes on the side of the otitic or intracranial disease; after the number it indicates the opposite side; before and after=bilateral changes. Doubling of the sign before or after the number denotes more marked changes in the eye of the correspond ing side.

A study of these records shows that the great majority of cases of intracranial otitic complications were examined at some time or another during the stay in the institution, for evidences of changes in the fundus oculi. A few cases were not so examined, either on account of death having come on suddenly a short time after admission, or because the classic symptoms of intracranial

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15. In addition to this material I have included all cases reported in the last (1906) volume of Transactions of the American Otological Society, of Intracranial Otitic Complications in which an ophthalmoscopic examination was made. They are one case of lateral sinus thrombosis (Gruening), and two cases of cerebellar abscess (Dench, A. Knapp). Gruening's case was particularly interesting as there was bilateral mastoiditis with exposure of both sinuses at operation. Characteristic temperature changes arousing suspicion of thrombosis, an examination of the fundus was made and hyperemia of the discs found on both sides. On only one side, however, did this progress to optic neuritis, and on the strength of this finding, and in spite of the absence of evident local changes in the sinus wall of this side, and the presence of some discoloration on the other, the apparently healthy sinus was opened and found to contain an infected clot. Here the diagnosis was made by the ophthalmoscope alone, not only unsupported, but actually contradicted by the local signs.

disease assured the diagnosis, or finally because the symptoms receded immediately after operation. These reasons are of practical weight, but from a scientific point of view it is to be regretted that systematic examinations of the fundi are not made in every case of operative ear disease. The presence of choked disc or of an optic neuritis several days after operation is open to several interpretations in regard to the period of development of the causal complication, whereas its significance is very much less ambiguous in case a normal fundus was found at a previous examination. Koerner lays stress, and justly, on the importance of repeated examinations "up to the time of cure or death, i. e., until the diagnosis has been established, etc." The corroboration of the ophthalmoscopic findings, or rather of the diagnosis which they are to assist, is unfortunately beset with more than usual difficulty. In the case of recovery the character of the ailment is often no more positively determined than it was before, or, at all events, there is no certainty of such degree as to enable us to stamp the diagnosis as agreeing with the intraocular changes or not. The findings at operation may be of value, especially in the event of positive evidence, in the form of the evacuation of pus, of a brain abscess, of an occluded, thickened or ulcerated sinus, discolored meninges or subdural abscess formation, but in other cases they fail to clinch the diagnosis. The only absolute verification, namely, by examination postmortem, is excluded, of course, in case of recovery, and frequently by the inability to obtain permission for autopsy. This is the rule rather than the exception, and unfortunately it is just in the most serious cases, those dying of cerebral complications, that an addition to our definite knowledge would be of greatest value. From this point of view our statistics, no matter how large, are much less instructive than the smaller ones of continental, and particularly of German, observers, who have been enabled in the great majority of cases to corroborate or disprove the intra vital clinical diagnosis by autopsy and pathologic examination postmortem. The postulate of examination by experienced and skilful ophthalmologists is fully met in the case of the institution cited in my study, as it has a large and active eye department, and numbers among its surgeons Gruening, Bull, Derby,[16] Callan, Weeks, Marple and other well-known oculists. The internes, too, have with few exceptions had a double service, with ample practice in the use of the ophthalmoscope and the study of fundus conditions both in the in- and out-patient department. In many instances the name of the examiner is mentioned.

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[16] July, 1907.

and in some, several consultants have expressed their opinions as to the fundus conditions. Where no examiner is specified the ophthalmoscopic findings were made by the house surgeon. In case of doubt or of unusual conditions the attending surgeons were called on for an opinion. As in many other reports of fundus conditions accompanying otitic complications, it appears that there is not sufficient attention paid to the necessity of a strict differentiation between optic neuritis and choked disc, or, in the case of already established atrophy, to the distinction between the simple and the postneuritic form. While in the latter case it may not always be easy even for the experienced ophthalmologist to detect slight traces of previous inflammation about an atrophic disc, such as irregularity of outline, proliferation or irregular distribution of pigment along the disc margin, filling up of the lamina cribrosa with connective tissue, irregularity of lumen, thickening of vessel walls or connective tissue striæ in their course, some or most of which changes may be wanting, there is usually no doubt at all, in a specific concrete case, whether we have to deal with neuritis or choked disc, at least in the early stage. And here, again, is a reason for early as well as for repeated ophthalmoscopic examinations; a choked disc may be engrafted or superadded to a neuritis, or *vice versa,* in a case, respectively, of an intracranial inflammation, such as meningitis being complicated by a process which greatly increases brain pressure, as, for example, brain abscess or internal hydrocephalus, or, on the other hand, in the still more unusual event of inflammatory changes in the meninges occurring in brain abscess or thrombosis.

The scope of this presentation does not permit of a detailed consideration of the pathogenesis and mechanical development of choked disc and optic neuritis, a subject of practical import for the interpretation of clinical pictures in cerebral disease, particularly as there is still a difference of opinion as to the exact manner in which these intraocular conditions are brought about, and a number of conflicting theories to explain them. We may in a general way accept the prevalent opinion that choked disc is due to embarrassment of venous off-flow from the globe due to pressure on the nerve either by sheaths distended with inflammatory fluid exudate, solid mass, or edematous transudate, or to edema of the nerve itself, as from neuritis, and is accordingly purely or mainly of mechanical origin, while neuritis may be due to circulatory disturbance, to bacterial infection, to the action of toxins working at a distance from a purulent focus, to mechanical compression as in choked disc, or to a combination of some or all of these factors. For the practical purposes of the otologist choked disc signifies a morbid process which has resulted in a compression of brain tissue or at least in diminution of free space and so to a rise in intracranial pressure, while optic neuritis points to an inflammation of or in the immediate neighborhood of the meninges and an extension of this process either by continuity, or more probably by circulating bacteria or toxins, to the sheaths, and eventually to the substance, of the optic nerve.

The two conditions are dissimilar in symptomatology, clinical course, and to a certain extent in prognosis. Choked disc is characterized by protrusion, often amounting to several diopters, marked edema, generally marked dilatation or tortuosity of the veins, with narrowed arteries. Retinal hemorrhages are not infrequent. The nerve fibers become swollen and the nerve head appears made up of radiating striæ, often flecked with linear hemorrhages and resembling a chrysanthemum. There is no marked irregularity or obscuration of the disc margin. In extreme cases, of course, the intense congestion may induce slight inflammatory changes so that there may be a mild neuritis in addition. After relief of excess pressure in the cerebrospinal fluid a marked degree of choked disc may entirely disappear. Atrophy is an unusual sequela and is usually attributable to complicating neuritis or to intercurrent disease of the nerve or descending degeneration or inflammation from central disease. In neuritis, there is usually little or no swelling of the disc, so that protrusion is not noted. Hyperemia, on the other hand, is marked, and the outlines of the disc soon become indistinct or entirely invisible. This in marked contrast to papillary stasis, in which the disc is often enormously swollen and prominent, yet distinctly visible. The obscuration of the disc in neuritis may be so marked that the position of the nerve head is merely indicated by or rather to be inferred from the converging vessels at the center of the fundus. Marked changes in the caliber or course of the vessels are infrequent, almost exceptional. On the other hand, we note changes in the vessel walls in the form of perivascular striations, connective tissue proliferation starting at the disc and accompanying veins and arteries out along the fundus, as well as deposits of fibrin in the physiological excavation of the disc, i. e., in the lamina cribrosa as well as at the margin. Of course, these changes are often obscured by the intense hyperemia and inflammation of the disc and by a fine opacity of the posterior layers of the vitreous and edema of the central part of the retina which almost invariably accompanies this condition. It is only in the later stages, particularly that of postneuritic atrophy, a frequent sequela, result, that these processes are made manifest by irregular outline of the disc, blocking up of the lamina and physiological excavation, and veiling of the markings of the cribrosa, perivascular connective tissue strands, and by pigment deposits on the disc or along its margins. Retinal hemorrhages are rare in uncomplicated optic neuritis. When present they may be due to choked disc, to vessel wall changes caused by mechanical obstruction of the circulation, or to vascular disease of a septic nature, as in pyemia or metastatic or septic retinitis.

In the construction of the table I have spoken, quite generally, of fundus changes, as opposed to the normal eyeground, in order to render the comparison of positive and negative findings more simple and graphic. In each case, however, the fundus conditions, whether neuritis or choked disc, and their distribution in one or other fundus or both, whether equally, or predominantly on one side, are indicated by the signs which are explained in the footnote. In the same table the cases are arranged in two main divisions, according as they resulted in death or recovery, so that it is possible to compare the frequency and severity of the fundus changes in mild and in fatal cases for every variety of intracranial complication. This, it seems to me, makes it much easier to get a general view of the subject than if we follow the example of Hansen and of Koerner and construct a number of tables dealing separately with the factors of distribution, severity, frequency and result. We are better able to judge of the prognostic significance of the presence or absence of fundus changes in endocranial complications if each form of disease is shown separately than if we make two categories, death and recovery, and compare the fundus findings in the two divisions for the entire number of cases. Instructive facts may be obscured by such figures which are actually out of place. For it is evident that figures which show a remarkably high percentage of fundus changes in fatal cases of, let us say, sinus thrombosis, will be balanced and lose their identity if combined with others showing indeterminate, negative or even opposite conditions in abscess or meningitis. The same considerations apply to a statistical study of the distribution of fundus changes, meaning the occurrence in one or other eye with greater frequency or severity, and the significance, if any, of bilateral changes.

A study of the individual and various forms of intracranial complication does not bear out, in my statistics, the conclusion of Koerner that the presence or absence of fundus changes is of no value in the prognosis of intracranial complications. The results as to distribution are also less ambiguous. Koerner found intraocular changes, without exception, in every instance, bilateral, and more often to a more marked degree on the sound than on the affected side.

EXTRADURAL ABSCESS.

In 6 cases of uncomplicated extradural abscess the fundus was invariably found normal. In 8 cases complicated with sinus thrombosis there were fundus changes in 3 (37.5 per cent.), all bilateral.

MENINGITIS.

In purulent leptomeningitis without other complication changes of the nerve head were found in 4 out of 5 cases, all of which ended fatally. The changes were bilateral in all but one case, in which they were limited to the side corresponding to the mastoid involvement. In this case, choked disc was found, which in another was associated with optic neuritis of the other eye. Of 9 complicated cases neuritis was found in 6, slight changes in the vessels in 1, and a normal fundus in but 2. The intraocular disturbance was without exception bilateral.

SINUS THROMBOSIS.

In sinus thrombosis fundus changes were found much more frequently than might have been expected from the reports of previous statistics. Of 17 uncomplicated cases, no less than 12 showed decided fundus changes. All the fatal cases, 12 in number, showed fundus changes, while the 5 without fundus changes all recovered. In these cases, too, the intraocular disturbance was always bilateral, with the exception of the case reported by Gruening and included in my statistics, already mentioned, and one other, in which a thrombosis of the central retinal vein, probably of metastatic origin, was the only fundus change. In sinus thrombosis with extradural or perisinus abscess, of 7 cases which recovered only 2 showed neuritis. Of the 2 fatal cases, one was normal, the other showing double neuritis. In all of this group, again, the fundus changes were bilateral, except in a case of general thrombosis of the sinuses complicated with meningitis on one side only, strange to say, and it was a question to which of the two intracranial complications the intraocular condition was to be attributed. The other cases of complication with meningitis are grouped under the latter heading above. Of the entire series of thrombosis cases, 28 in number, changes were found in 17 (61 per cent.). This observation is in marked contradiction with the figures of Hansen, who found them in only 37 per cent. of his thrombosis cases, and of Koerner, who had 5 uncomplicated cases, all but one with normal fundus. In view of these figures we must modify Koerner's conclusion that unilateral occlusion of the venous channels in lateral sinus and jugular does not seem sufficient in most cases to cause congestion in the ocular fundus.

BRAIN ABSCESS.

One case of uncomplicated temporo-sphenoidal abscess and 3 of uncomplicated cerebral abscess showed normal conditions in the fundus. In one case of uncomplicated brain abscess there was neuritis. Reviewing the combination of observations thus briefly reported, we may still say that changes in the fundus oculi may be absent in all and any forms of endocranial affection, but, and this is almost as unfortunate from the standpoint of differential diagnostics, they may also be present in any and all, with the possible exception of uncomplicated extradural abscess. An exclusive or more markedly unilateral ocular involvement is rare and generally without significance, but in sinus thrombosis it may be a valuable sign. The prognostic significance of fundus changes for the course of the intracranial complication is somewhat obscured by the fact that mild cases are often not examined ophthalmoscopically, while in the fatal cases the condition of the fundus is more apt to have been investigated at some time or another. It would appear that the absence of neuritis in thrombosis is of good omen, as it was not missed in a single one of my fatal cases, but the presence of neuritis does not make the outlook more grave, as there is about an equal percentage of recovery and of death after it has been found.

Dr. J. D. Richards, of New York, has been kind enough to give me the results of his own experience in this field. He has seen extradural abscess produce papillitis in three cases, in all of which it was on the affected side. What percentage this is he is unable to state, but thinks it must be exceedingly small. In sinus thrombosis it is probably not over 5 per cent. Richards has not seen the high percentage referred to by some writers as associated with thrombosis. A point of interest in the sinus cases to which Jansen and he have called attention is that those cases which showed neuro-retinitis had the jugular bulb thrombosed. In these cases the inferior petrosal is also blocked and the damming back of venous blood upon the cavernous sinus and thus upon the ophthalmic vein and intraocular vessels is probably responsible. He believes that when we have a sufficient number of observations to enable us to draw definite conclusions, this agency of jugular bulb thrombosis in the causation of fundus changes will be corroborated. At least three of the cases collected by me seem to support this view. In one (43) there was a thrombus in the lower part of the sinus only, probably implicating the bulb, and double optic neuritis. In another (67), ending fatally, there was neuritis on the thrombosed side and later positive evidence of cavernous sinus involvement in the form of edema of the eyelids, beginning on the affected side and afterwards involving both eyes. In a third case (56), the sinus was apparently normal at operation. A peaked temperature curve, pressure pain along the jugular, and slight neuritis, more marked on the affected side, led to evacuation of the sinus and excision of the jugular vein, followed by recovery. In this case it would seem that there was a primary thrombosis of the vein, which at the time the mastoid was opened had not yet involved the upper part of the sinus. The conditions here were almost paralleled in Gruening's case, which I have included in my statistics, as the fundus changes were found only on the affected side, and the sinus appeared healthy not only at the primary operation, but at the time of jugular extirpation. On incision, however, it was found to contain an infected clot, so that here, too, it seems quite probable that there was a primary bulb thrombosis with very little tendency to advance upwards, but with rapid infectious softening.

Comparing the results of his own investigation with those of Hansen, Sessons and Koerner, the writer has but little to add, except in corroboration. The absence of fundus changes in a given case has no significance. Its presence, on the other hand, is of the greatest importance. This bearing is not strictly diagnostic, as we can not draw any conclusion from the presence of fundus changes as to the nature of the intracranial affection, but it is an absolute indication of the presence of some complication, and as such makes it advisable, if not imperative, to explore the cranial cavity and, if possible, remove the source of suppuration and of local as well as systemic infection. To put the case in a nutshell, fundus changes may be absent in intracranial complications, but intracranial complications are never absent with fundus changes. The otologist should make his own ophthalmoscopic examinations. This does not imply that an ophthalmologist is not to be consulted in a doubtful case and the observations compared. Early and repeated examinations in each and every case can only be assured by the ability of the aural surgeon to use the ophthalmoscope with skill and experience. Ophthalmoscopic examination is usually neglected unless the previous history or the development of other symptoms of intracranial involvement directly suggest it. Early and repeated examinations would add greatly to the value of our statistics and to our knowledge of the pathogenesis and significance of fundus changes, and routine examination on admission of all operative cases would not only supply much valuable material, but avoid the loss of important observations due to the frequent impracticability of carrying out an ophthalmoscopic examination on the sudden development of ominous symptoms, the preparation for further urgent operation, or, indeed, sudden death.

In addition to the observations on fundus changes, I wish to report quite briefly on one or two other ocular symptoms. In a comparatively large number of cases of generally uncomplicated otitis or mastoiditis, edema of the eyelids limited to the affected side was found as an expression indicative of a marked involvement of the zygoma cells (1, 9, 15, 29), complicated in 2 cases (9, 29) by subperiosteal abscess. The same symptom was found in 3 cases of secondary inflammatory infection of the soft parts, after removal of all diseased bone (25, 26, 29) in ordinary furunculosis of the external auditory canal (14, 63) and in stitch-hole abscess developing a week or two after operation (10, 23). In two cases, finally, this edema of the lids was due to conditions which had no connection or only an indirect connection with the otitis, as erysipelas (71) and frontal sinusitis (11). Lid edema as a symptom of intracranial complication was observed in 3 cases. Once in spontaneous recovery from a probably incomplete sinus thrombosis (21), again bilaterally in sigmoid sinus thrombosis with thrombosis or stasis in the cavernous sinus (67). Ophthalmoplegia occurred in 2 cases of brain abscess (2, 19), and, again, as an expression of mechanical fixation of the globe by inflammatory products in the orbit, in thrombosis of the cavernous sinus (38). Convergent strabismus was noted in meningitis (72), meningitis with sinus thrombosis (64), and meningitis with epidural abscess (32, 57, 59). Abducens paralysis occurred in one case of uncomplicated (?) middle ear suppuration, and once, probably as a result of traumatism, after exploratory incision of the brain (13), once in combination with paralysis of the trochlearis in a case of brain abscess with meningitis (30), and in one case, finally, of meningism following sinus thrombosis (55). Conjugate deviation of the eyes, as well as nystagmus, was found in several cases of brain abscess and meningitis, and in one of lateral sinus thrombosis, but, as the case histories and autopsy records, respectively, of these cases did not completely explain the symptoms or establish the diagnosis on a pathologic-anatomical basis, they will not be considered further.

A PRELIMINARY STATISTICAL INQUIRY INTO THE REFRACTIVE AND SOME PATHOLOGICAL CONDITIONS OF THE EYES OF FIVE HUNDRED MEN ABOVE SIXTY YEARS OF AGE

IN THE NATIONAL MILITARY HOME, NEAR DAYTON, OHIO, EXAMINED UNDER HOMATROPIN, TOGETHER WITH FOUR HUNDRED AND FIFTY-FIVE BLOOD PRESSURE RECORDS.

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This paper is largely a statistical inquiry into the eye conditions and blood pressure records of 500 men over 60 years of age, with an average age of 70.76 years, inmates of the Central Branch of the National Military Home, near Dayton, Ohio, of which I have been a medical officer for twenty-three years.

This branch cared for 6,974 men during the year ending June 30, 1906. Of this number only 435 were under 60 years of age; the average age of the whole number was 67 years. There were 475 deaths, the average age of decedents being 70.83 years. The ratio of deaths per thousand was 63.11. It is not believed that a like number of men can be found anywhere, considering physical condition, age, uniform surroundings and under such control that an examination of the kind to be reported can be made. Hence the opportunity is exceptional. Because disability of some kind must be shown to entitle a man to admission to the Home the average physical condition of the inmates is not good. They were recruited, as a rule, from the middle and lower laboring classes, which from 1861 to 1865 made up the bulk of our rural population. The hard lines of laborious occupations and vices have left their impress on the constitutions of the men whose vitality had already been sapped by the stern realities of war, gunshot wounds, poor and insufficient food at times and exposures of all kinds. From the class of material described 500 complete examinations have been made. By complete I do not mean that I have gone exhaustively into all possible intraocular conditions and constitutional states. The paper does not contemplate this. The purpose is to show by abundant statistics what pathological eye conditions have been observed in a large number of old men, and their proportion to the total number, rather than an attempt to define all the pathological conditions on which the disease states have depended or to attempt to reach conclusions concerning them from the blood pressure records. The writer regrets that it has not been possible to have a careful physical examination of the heart and an exhaustive examination of the urine made in every case, and also that in all cases with a blood pressure above 160 a record was not made as to the depth of the anterior chamber, tension of the globe, etc. The association of these last-named conditions with glaucoma is generally admitted, but further study as to the rôle played by arterial sclerosis with high blood pressure in its etiology is desirable.

The men examined have not been selected; most of them have been taken as they came to the clinic for eye and ear treatment from the camp. These represent the best type of men physically that we have. A large number have been sent in by the assistant surgeons from the main hospital and out-wards. These represent the worst type of men, from a physical standpoint, as they are all hospital patients, suffering from all manner of diseases and disabilities. The two classes, however, will give a fair average of the physical condition of the inmates. While no selection of cases has been made, certain men have been rejected as not fit subjects for the examination for the following reasons:

1. Not having attained to 60 years of age.
2. A want of sufficient intelligence to appreciate what was being done, and inability to answer the questions asked. Some have rejected themselves by refusing to have the mydriatic instilled.
3. No case has been accepted unless a satisfactory view of the periphery of the lens, the vessels, and the details of the fundus could be seen in one or both eyes.

My personal attention has been given to the details of every examination, except blood pressure records, which have been taken in the vast majority of cases by my assistant, Dr. H. A. Slusser, of the medical staff of the Home.

All blood pressure records presented in these statistics have been taken with the Janeway instrument, with 12 cm. cuff and between 2 and 5 o'clock in the afternoon, when the light mid-day meal had passed out of the stomach and intestinal digestion was well advanced. It is not believed that the increase in pressure (from 10 to 30 degrees) often observed after a hearty meal will obtain in these men whose diet, while nutritious and wholesome, is simple and plain. Every test has been made on the left arm when possible, and in the sitting posture in every case, the arm on the level with the heart. Two or three tests have usually been made and the reappearance of the radial pulse has been the basis from which systolic pressure has been read; diastolic pressure has also been taken regularly, but it has little additional value in these records, which have been taken for diagnostic, not for therapeutic purposes. Four hundred and fifty-five records have been taken with an average systolic pressure of 163.6, and, while they have only been taken once, it is not believed that this will materially lessen their value, especially in the (pathological) cases with a pressure of 160 and upward. Continued high pressure can never exist without hypertrophy of the heart which, when once established, is constant and is not subject to wide fluctuations from day to day, so that for our purpose one examination would seem to be sufficient. It only falls when dilatation and broken compensation are reached.

Among those who use the sphygmomanometer there is not as yet complete agreement on what may be considered normal blood pressure; age, sex, occupation and other factors influence the height of the blood pressure in normal individuals. Therefore, we can at present speak only of certain lower and upper limits, which will be generally accepted as normal. Just beyond these limits will lie a debatable territory, considered normal by some observers, or for particular individuals, abnormal by others or for other individuals. This is particularly true of the pressures between 145 and 160 M/Hg when found in elderly people, since it will always be difficult to separate the effects of age *per se* from the effects of concomitant pathological changes in the cardio-vascular system. In this paper I shall take 130 M/Hg as the normal standard, because more in harmony with the general physical condition of these men, none of whom were called upon for either bodily or mental activity, rather than the 145 M/Hg which Janeway makes the upper limit of normal systolic pressure. It should be remembered that we have examined different classes of men. It is evident that the age at which these pressures were found had much to do with their clinical significance; in other words, a pressure of 130 in a young man, 20 years of age, if constant, would have far more clinical significance than the same pressure in a man 60 years of age. There seems to be no upper limit of pathological pressure, except the amount which the arteries will stand; I have not found it above 280 M/Hg in this series of cases.

A large proportion of these statistics will be given in connection with blood pressure records, the purpose being to show the relation or association, *if any,* of arterial sclerosis and hypertension to the intraocular conditions or diseases to which so large a proportion of these men are subject. It will, perhaps, conduce to a clearer understanding of the subject, if I depart for the moment from the statistical plan of the paper and give briefly my conception of the rôle arterial sclerosis and resulting hypertension play in the causation of the ocular diseases so often found in this class of men.

The blood pressure may vary from 50 M/Hg in profound shock to 95 M/Hg, which is about the systolic pressure required to maintain the circulation in health, up to a pressure of 300 M/Hg or even more in disease. These upper limits of blood pressure for self-evident reasons are not so definitely fixed as are the lower pressures, and the power to withstand the high pressure depends on the condition of the arterial walls; these conditions explain the saying that "A man is as old as his arteries," and they also explain why certain families are long lived, attaining to the eighties in good physical and mental condition, while other families age early and are old in the forties, being frequently subjects of death from cerebral and other hemorrhages.

The blood supply of the eye, considering its importance, seems comparatively limited. Only three small arteries, terminal branches of the ophthalmic, enter it. Three of its most important component parts, the cornea, lens and vitreous, have no direct blood supply, but are nourished at second hand, the first two by the anterior and posterior ciliary arteries and the last through the uvea and the retina by its own artery. Obviously any disease which narrows the caliber of these vessels or reduces the elasticity of their walls will affect the circulation of the blood through them and the nutrition of the structures they supply, and degenerative changes result. If we leave the realm of theory and study what we really see in such cases, we find that what is predicated in these statements really takes place in disease. Witness the more than 58 per cent. of cataractous lenses, the per cent. of vitreous opacities, pressure on the veins and chorioidoretinal changes at the disc, which have been observed. We are so accustomed to observing the effects of hyperplastic inflammatory processes that we are prone to overlook the very important classes that are directly degenerative in character, as we see them in the cornea, lens, vitreous, chorioid and retina. I shall not attempt to go into the pathology and etiology of the arteriosclerosis; these are outside the scope of the paper. These and many other statistics show that arteriosclerosis is of frequent occurrence in elderly people. If it is not found, it is because it is not looked for. These statistics show that of 455 men whose systolic blood pressure has been carefully taken, only 15, or 3.4 per cent., had normal blood pressure, that is under 130 M/Hg, according to the standard used in this paper. (It has been suggested to me that the standard is too low for this class of men, whose average age is 70.76 years, and that a higher normal limit of 145 M/Hg would be nearer correct for them. Having already given the reasons for adopting the lower standard, 130 M/Hg, I may add that 112 men, or 24.5 per cent., gave a reading of 145 M/Hg and under.) The remaining 438, or 75.5 per cent., comprise the debatable pressure (130 to 160 M/Hg), and the distinctly pathological pressure (160 to 280 M/Hg). von Schultin[1] has shown that "the maximum pressure in the ophthalmic artery is only 2 to 15 M/Hg less than that of the aorta itself." Experience has shown that the diffused form of arterial sclerosis, or, as it is sometimes called, angiosclerosis or arterio-capillary fibrosis, attacks by preference the small arteries both superficial and visceral. Its predilection for splanchnic vessels and the terminal arteries in the important organs above the diaphragm and its influence in the production of high blood pressure when so located are well known. This predilection also explains why the condition is so often found in the retinal artery where it is so open to inspection that the clinical picture, tortuous macular arteries, pressure on veins at crossings, ampulliform dilatations, silver wire arteries and hemorrhages, is familiar to all oculists. These conditions were often the first indications of the presence of the disease elsewhere throughout the body before the sphygmomanometer came into use. The value of the information furnished by this instrument in connection with the ophthalmoscope can not be overestimated. It is so important and far reaching in the information it gives that no examination of an individual beyond 50 years of age, so far as diagnosis, and particularly prognosis, are concerned, is complete without one or more blood pressure records. Recent studies along the line of blood pressure and arterial disease seem to indicate that certain ocular complications of several well-known general diseases are manifestations of the arterial disease so often associated with them. This is particularly true of the eye conditions found in those forms of Bright's disease which are now regarded by many pathologists as renal phases of arteriosclerosis, and not as primary diseases of the kidneys. It is of interest to note that Bright himself, as early as 1836, had substantially this modern idea (in a very crude form) of the disease which bears his name.

"The normal strain imposed on the arterial walls by intra-arterial pressure of the blood is of considerable magnitude, amounting to 156 grams for every square centimeter of surface, about 2¼ lbs. per square inch."[2]

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[1] Janeway, Clinical Study of Blood Pressure, p. 30.

All the examinations have been made as follows:

The name, regiment and company; the nationality, age, occupation, drinking habits; specific history has been asked for in every case, etc.

Nationality has been taken in 494 cases and is shown by statistics as follows: Three hundred and twenty-eight, or 65.6 per cent., were Americans; 59, or 11.8 per cent., were Germans; 85, or 17 per cent., were Irish; 12, or 2.4 per cent., were English; 6, or 1.2 per cent., were not recorded; 1, or two-tenths of 1 per cent., was a Swiss; 1, or two-tenths of 1 per cent., a Swede; 1, or two-tenths of 1 per cent., was an Austrian; 2, or four-tenths of 1 per cent., were French; 4, or eight-tenths of 1 per cent., were Canadians, and 1, or two-tenths of 1 per cent., was a Welshman.

Of the 290 men with cataract in some stage of development, 187, or 64.5 per cent., were Americans; 35, or 12.7 per cent., were Germans; 52, or 18 per cent., were Irish; 2, or seven-tenths of 1 per cent., were French, and of the remaining 14 men I have not figured the nationality.

In fixing the lower limit of age for the examination at 60 years, I have had in mind the limit which would furnish the largest number of cases, and in which so-called senile changes, which constitutes an interesting and perhaps debatable part of the study, begin to manifest themselves with considerable regularity, or at least should be looked for. It seems to me wrong, and at variance with the facts, to call certain degenerative changes in the eye structures *senile,* if by that we mean that they are normal or physiological results of age. This is particularly true of fluidity of the vitreous, incipient nuclear and cortical opacities of the lens and chorioido-retinal disturbances around the disc so often seen in elderly persons. The essential element in these latter cases is atrophy of structure and disturbance of the pigment layer. Exactly the same appearance is seen in well-recognized types of chorioiditis and chorioido-retinitis. Undoubtedly the first is as distinctly pathological as the last condition. While it is natural for all persons to age or become senile, using the word in its generally accepted meaning, the writer believes that the connective tissue proliferation which preceded or brings about these senile changes is distinctly pathological.

Of the first 100 men, the average age is 67.73 years; of the second 100, 79.72; of the third 100, 69.09; of the fourth 100, 68.56, and the fifth 100, 68.70. The average age of the 500 men is 70.76 years. Of 447 men whose blood pressure in relation to age I have figures, only 15, or 3.4 per cent., had pressure under 130; 160, or 33.6 per cent., had pressure under 160 M/Hg, and 281 men, or 60 per cent., had a pressure of 160 to 280 M/Hg. Nineteeen men 60 years of age had an average pressure of 166.6 M/Hg; 34 men 64 years of age had an average of 158.2; 20 men 68 years of age had an average of 170.3; 17 men 72 years of age had an average of 171.5; 10 men 76 years of age had an average of 170; 7 men 80 years of age had an average of 180. The average for the whole number was 163.6. I have purposely taken the average in each fourth year for the sake of brevity. Above 80 years of age the number in each year is so small that the averages would be high or misleading. For example, 4 men at 82 years of age gave an average of 187; 2 men at 84 gave an average of only 165; 2 men at 90 gave an average of 205, and 1 man who claimed to be 101 years old had a pressure of 200. The highest pressure recorded, 280, was observed in an Irishman, 75 years old, who had been a hard laborer, a regular drinker, had a specific history and marked hypertrophy of the heart. In the first 100 cases, the 10 highest systolic pressures averaged 207.2, and the second 100, 204; in the third 100, 212; in the fourth 100, 223, and in the fifth 100, 199. Average for the 50 highest systolic pressure, 206.6; average for the 50 lowest diastolic pressure, 77.

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[2] Nichols: Washington Medical Annals, July, 1906.

OCCUPATIONS.

These represent about 50 different kinds of employment, too many for a statistical table. I have grouped them under two classes. In the first class of 371 men who had followed hard and laborious callings, of which the farmer and day laborer are a type, only 123, or 33.15 per cent., had blood pressure less than 160 M/Hg, while 248, or 66.85 per cent., had pressure from 160 to 280. In the second class, of 142 who stated that they had followed professions, occupations and trades, of which the physician, clerk and bookkeeper may be taken as a type, 65, or 45.8 per cent., had blood pressure of less than 160, and 77, or 54.2 per cent., had pressure above 160. In the first class, 66.85 per cent. had pressure above 160, while in the second class 55 per cent. had pressure above 160. It is believed that the classification referred to above is a satisfactory one and that it demonstrates that the kind of a life a man has lived during his productive working years has more influence in determining what his physical condition and that of his arteries will be at 60 years of age. for example, than the number of years he has lived. Vital statistics confirm these statements. It is of record that the married man, by a better regulated life, has an expectancy of 60 years, while an unmarried man, for the opposite reason, has an expectancy of only 45 years.

Drinking habits have been inquired into in every case and are represented statistically as follows:

Of 500 men, 89, or 17.8 per cent. claimed to be temperate; 86, or 17.2 per cent., stated that they were regular drinkers; 45, or 9 per cent., stated that they drank periodically, while 240, or 48 per cent., claimed to be moderate drinkers, and in 40, or 8 per cent., no record was made. Of 86 men who acknowledged being regular drinkers, 55, or 64 per cent., had a blood pressure of 160 or over; of 226 men who claimed to be moderate drinkers, 151, or 66.8 per cent., had a blood pressure of 160 and over; of 44 men who claimed to be periodical drinkers, 23, or 52 per cent., had a blood pressure of 160 and over; of 92 men who claimed to be temperate, 57, or 62 per cent., had a blood pressure of 160 and over. These and other statistics show that the influence of alcoholic drinks in the causation of arterial disease has probably been overestimated, and that as an etiological factor it does not compare with tobacco when used to excess.[3]

SPECIFIC HISTORY.

Admitted by 34 men, or 8.2 per cent., denied by 420, or 48.42 per cent. No record obtained in 36, or 7.8 per cent. Of the 34 men who gave a specific history, none showed blood pressure of less than 130, which, I repeat, is the upper limit of normal pressure according to the standard used in these statistics. Eight, or 23.5 per cent., showed a pressure under 160, averaging 143, and 26, or 76.4 per cent, showed pressure above 160, averaging 185 for the whole number.

It will be observed that these 26 men gave a higher average of blood pressure than any class presented, the six highest giving an average pressure of 237.5. These statistics are reliable as far as they go, because the men themselves have admitted the infection and the sphygmomanometer has confirmed our understanding of the high blood pressure to be expected in such cases, but the motive for denying infection is too strong for the average man to resist; hence the number of non-syphilitics is too large. Diseases due to vicious habits are not pensionable. The average soldier understands this and fears that by admitting a specific infection he may in some way jeopardize the pension he is receiving. Here is the chief motive for denying infection.

The following statistics showing the relationship of age, blood pressure and lens opacity is one of the most, if not the most, important set of the series.

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[3] Janeway: Clinical Study of Blood Pressure, p. 119.

Of 910 eyes conforming to the requirements of the examination, 529, or 58.1 per cent., had some kind or degree of lens opacity. Of 25 of these, I have no blood pressure record, so that the ratios are on a basis of 504 eyes, *not men.* Only 16, or 3.2 per cent., had a blood pressure of less than 130, the average was 118.2. The average age of the 16 was 67.3 years. One hundred and fifty-three, or 30.3 per cent., had a blood pressure between 130 and 160, the average was 138.9. The average age of the 153 was 68.2 years. Three hundred and thirty-two, or 66.5 per cent., practically two-thirds, had blood pressure ranging from 160 to 280, the average of which was 179.7. The average age of these 332 was 76.1 years. Fifty, or 9.9 per cent., of the 504 were classed as mature cataracts; 102, or 20.2 per cent., as incipient cortico nuclear; 255, or 50.6 per cent., as incipient cortical; 84, or 16.7 per cent., as incipient nuclear; 7, or 1.4 per cent., as postpolar; 4, or eight-tenths of 1 per cent., as capsular, and 2, or four-tenths of 1 per cent., as traumatic.

INCIPIENT CORTICAL CATARACT.

The 255 eyes with incipient cortical cataract had an average age of 69.4 years, ranging from 60 to 95. The blood pressure ranged from 110 to 250, averaging 163.8; 15, or 5.9 per cent., had blood pressure below 130, the average being 117.7. The ages varied from 62 to 74, the average being 67.5. Eighty-one, or 31.8 per cent., had blood pressure between 130 and 160, which averaged 137. The average age was 67.8; the youngest being 61 and the oldest 85. There were 159, or 62.3 per cent., with blood pressure of 160 or over, the average being 177.5. Their ages, from 60 to 95, averaged 67.8 years. Eighty-five, between 60 and 65 years old, with an average age of 64.3, had an average blood pressure of 160.4, ranging from 110 to 250. Eighty between 65 and 70, with an average age of 68.4, had an average blood pressure of 163.5, ranging from 125 to 230. Thirty-eight between the ages of 70 and 75, with an average age of 73.2, had blood pressure ranging from 130 to 220, with an average of 181.8. Twenty-one between 75 and 80, with an average of 181.78, had blood pressure from 130 to 210, which averaged 174.5. Thirteen between 80 and 85 years, with an average of 83.5, had blood pressure ranging from 150 to 175, which averaged 162.3. Two at 86 years of age had blood pressure of 190. Six between 90 and 95, with an average age of 94.3 years, had blood pressure ranging from 160 to 180, which averaged 168.3.

In every one of this class the opacity showed first in the lower half of the periphery, with a marked tendency toward the inner quadrant. Mittendorf[4] has suggested that accommodation and convergence have much to do with the uniformity with which this occurs, but it seems more reasonable from what we know of the pathology of incipient cortical cataract to think that gravity of the fluid portion of the degenerating lens structure offers a better explanation of its uniform location in this part of the lens.

INCIPIENT NUCLEAR CATARACT.

Of the 84 eyes with incipient nuclear cataract, the average age was 72 years, the ages ranging between 61 and 90. The blood pressure was between 130, the lowest, and 250, the highest, averaging 169.7. Twenty-two, or 26.2 per cent., had blood pressure between 130 and 160, averaging 139. The ages of these were between 61 and 78 and averaged 68.8 years. Sixty-two, or 73.8 per cent., had blood pressure of 160 and over, which averaged 192.9. The ages of these, between 64 and 90, averaged 77.9 years. There was none with blood pressure below 130. There were ten below 65 years of age, with an average age of 63, and average blood pressure of 164. Thirty-four between 65 and 70 averaged in age 64.8 years and had blood pressure which averaged 171.2. Fifteen between 70 and 75 averaged 73.3 years and blood pressure of 163. Eighteen between 75 and 80 averaged 77.9 years, with blood pressure of 169.2. One, aged 85, had blood pressure of 170. Six between 85 and 90 averaged 89 years and had blood pressure which averaged 190.

A condition that has been observed with considerable regularity when the cortex has been free from spokes and striæ has been a more or less marked definition of the nucleus, which has varied from a thin nebulous opacity to an apparently dense opacity which, when looked at obliquely and silhouetted against the red background of the fundus, has presented a striking picture of apparent cataract. This increase in the index of refraction of the different layers of the lens is well understood and is consistent with normal vision; it only deserves to be emphasized in order that it may not be mistaken for the more marked condition of incipient nuclear cataract. The condition described by Halben[5] under the title "Apparent Cataract." has not been observed.

INCIPIENT CORTICO NUCLEAR CATARACT.

In 102 eyes with incipient cortico nuclear cataract had an average age of 71.9 years, ranging from 60 to 95. They had a blood pressure ranging from 160 to 280, with an average of 168.5. Of these, 30, or 29.4 per cent., had a blood pressure between 130 and 160, with an average of 142.2. Their ages ranged between 62 and

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[4] Transactions of the American Ophthalmological Society.
[5] von Graefe's Archiv. für Ophthalmologie, vol. lxii.

85, averaged 70.9 years; 72, or 70.6 per cent., had blood pressure between 160 and 280, which averaged 179.8. The ages of these, between 60 and 87, averaged 77 years. Thirty-two, or 32 per cent., had an average age of 63.5 and blood pressure ranging from 160 to 200, with an average of 167.2. Twenty-one between 65 and 70 had an average age of 68, and blood pressure ranging from 150 to 210, which averaged 170. Sixteen with ages varying from 70 to 75 had an average age of 72.7, and blood pressure, ranging from 135 to 280, averaged 170. Fourteen between ages of 75 and 80 averaged 78.4. The average blood pressure was 166, the lowest being 135 and the highest 200. Fifteen between the ages of 80 and 85, with an average of 83, had blood pressure ranging from 145 to 200, averaging 172.3. Two with blood pressure of 170 were 87 years of age, and 2 at 95 years of age had a blood pressure of 200.

POSTPOLAR CATARACT.

Of 7 eyes with postpolar cataract, the average age was 68.1 years and the blood pressure was 161.4, ranging from 140 to 180. Four below 160 had blood pressure averaging 150, and their ages ranged between 60 and 70, averaged 65.8 years. Three with blood pressure 160 and over, averaged 176.7, while their ages, 68 and 75, averaged 72.7 years.

TRAUMATIC CATARACT.

The two traumatic cataracts were aged 65 and 71 years, respectively, and had blood pressure of 165 to 185.

CAPSULAR CATARACT.

The four capsular cataracts were aged between 65 and 79 and averaged 70.3 years. Their blood pressure ranged from 125 to 180 and averaged 154. One had blood pressure of 125 at 65 years of age. One had blood pressure of 140 at 70 years. Two had blood pressure of 170 and 180 at ages of 67 and 79 years, respectively.

Of 25 cataracts there is no record of blood pressure; their average age was 74. They were classified as follows: 14, incipient cortical; 5, incipient cortico-nuclear; 4, incipient nuclear; 1, mature, and 1, post-polar.

MATURE CATARACT.

Of 50 cases the average age was 68.5 years, ranging between 60 and 85. The blood pressure was 161.3, ranging between 130 and 210. Fifteen, or 30 per cent., of these had a blood pressure between 130 and 160, averaging 139.3. The average age of these 15 was 66.4 years. Thirty-five, or 70 per cent., had a blood pressure between 160 and 210, averaging 170.7. Their ages averaged 69 years. Twenty of these 50 were between 60 and 65 years of age, with an average of 63.1 years. Their blood pressure ranged from 130 to 200 and averaged 160. Fifteen were between 65 and 70, with an average of 68 years, and blood pressure from 130 to 185, averaging 162.3. Nine were between 70 and 75, having an average of 72.2 years, their blood pressure ranging between 145 and 200, averaging 169.5. Three with an average age of 77, between 75 and 80, had a blood pressure of 178.3, and three at 85 had blood pressure of 190. None of these had a blood pressure below 130.

Four hundred and forty-one men, or 82.2 per cent., showed some degree of chorioido-retinal disturbance around the disc; 140, or 31.7 per cent., had blood pressure below 160 and 301, or 68.3 per cent., had pressure above 160. Of 283, or 64 per cent., in whom the condition was noted, 249, or 56.5 per cent., had the atrophic and 34, or 6.8 per cent., the pigmented crescent of chorioidal disturbance; 46, or 9.2 per cent., had chorioidal rings; 43, or 8.6 per cent., had the atrophic and 3, or six-tenths of 1 per cent., had the pigmented ring of chorioidal disturbance; both crescents and rings have been limited to 3 millimeters or less in diameter; larger areas have usually been associated with the disseminated form of chorioiditis and are grouped under it.

Tortuous retinal arteries were observed in 25 cases, 5 per cent.; none of these had blood pressure of less than 130, and there were 5 between 130 and 160 and 20 with pressure of 160 and upward. Of these, two had 170, three had 180, four had 190, and five had 200 M/Hg pressure.

Forty-one cases, or 8.2 per cent., showed some degree of compression of the retinal veins where the hard arteries crossed them. These degrees of compression varied from slight indentation to well-marked ampulliform dilatation and hemorrhages. None of these had a blood pressure under 130; 6 showed from 130 to 160, and 35 ranged from 160 to 280. Of these, two had 160, four had 170, five had 180, four had 190, eight had 200, and two had 230 M/Hg pressure.

Papillitis and hemorrhages were observed five times, or 1 per cent., and four of these had a blood pressure above 160 as follows: one at 160, one at 185, and two at 200.

White lines along the blood vessels (perivasculitis) were observed six times, once with pressure of 150 and once, each, with 160, 170, 180, 185, 210. No well-marked cases of silver wire arteries has been observed, and two cases of cilio-retinal arteries have been seen.

Of 500 men with 910 eyes, the following records of refraction were obtained:

Fifty-four, or 5.5 per cent., were emmetropic; 507, or 55.7 per cent., to which can be added 21, or 2.3 per cent. of vision after cataract extraction, making a total of 528, or 58 per cent., hypermetropia; 30, or 3.3 per cent., had hypermetropia and hypermetropic astigmatism; 10, or nine-tenths of 1 per cent., had simple hypermetropic astigmia.

Forty, or 4 per cent., were myopic. Three, or four-tenths of 1 per cent., had myopia and myopic astigmia. Two, or two-tenths of 1 per cent., had simple myopic astigmia, and in 33, or 33.2 per cent., vision was not improved with glasses.

Three hundred and eighty, or 77.4 per cent., stated that they had never worn glasses constantly. Forty-one, or 9.1 per cent., had worn glasses constantly, and of 72, or 14.4 per cent., I have no record. Of 274 men with some kind or degree of lens opacity or vision after cataract operation in relation to refractive conditions and their correction by glasses, 162, or 65.6 per cent., were hypermetropes; 116 of these had never worn glasses constantly; 19 had worn them constantly, and of 27 there is no record. Twenty-one, or 7.7 per cent., were myopic. Of these, five had worn glasses constantly, 13 had never done so, and of three there is no record. In 83, or 30.3 per cent., vision was not improved by glasses; 71 of these had never worn them constantly, one had done so, and of 11 there is no record, and 8 with emmetropic eyes showed incipient cortical opacity in the extreme periphery of the lens.

COLOR OF IRIDES.

In this classification no variation in shades of color are considered; all shades of blue are classed in blue, the same with gray, brown, hazel and black. Fifteen men, or 3 per cent., had black eyes; 173, or 34.6 per cent., had blue eyes; 150, or 30.1 per cent., had gray eyes; 96, or 19.2 per cent., had hazel eyes, and 66, or 13 per cent., had brown eyes.

The average pupillary distance of the 500 men, measured with a small millimeter rule, was found to be 6.2 centimeters, about 2 7/16 inches. The transverse diameter of the cornea, usually of the left eye, had been measured with the same millimeter rule. The average diameter had been 12 millimeters. This perhaps is slightly larger than the average. It should be remembered, however, that the soldiers of the civil war were, as a rule, larger men than the average in civil life in that day, for the reason that the standard of admission to the army eliminated undersized men. The average size of the pupil has been 3.3 millimeters. As a rule, between 60 and 70 years of age the degree of dilatation from 2 to 3 drops of a 1 per cent. solution of homatropin in each eye has been sufficient for the purpose, and has about equaled twice the size of the pupil normal for the individual, on an average. For example, if the pupil was 3.5 mm. in diameter, the degree of dilatation has been about equal to 7 mm. After 70, the pinhead pupil so commonly seen will not dilate to double its natural size, a 2 mm. pupil will average 4, a 3 mm. pupil will average 5 and occasionally 6 mm. There has also been observed a close relation between the diameter of the cornea, the size of the pupil and the degree of dilatation to be expected. No case of glaucoma has developed from the use of the mydriatic in these 910 eyes.

My thanks are due Major F. W. Roush, Surgeon of the Home, for permission to conduct this inquiry. His sympathy and cooperation in the work is respectfully acknowledged.

For the aid and assistance given in the preparation of these statistical tables, Dr. J. W. Millette, my assistant in St. Elizabeth Hospital, has my sincere thanks.

As has already been stated, most of these blood pressure records have been made by Dr. H. A. Slusser. I can vouch for the substantial correctness of his readings, and it is a source of great pleasure to testify to his zeal and untiring interest in the prosecution of the study.

I am under many obligations to Dr. T. C. Janeway, of New York, for his kindness in looking over the manuscript and for two important corrections in the subject matter which have made it clearer and more comprehensive in relation to blood pressure. But it is not intended to imply that he assumes any responsibility for any of the statistics or the statements made, by having done so.

Dr. W. J. Conklin, of this city, has given me much valuable aid, for which my thanks are due.

DISCUSSION.

Dr. Jackson, Denver, Colo.:—Through the kindness of Dr. Greene I had an opportunity of looking over his statistics, and the thing that struck me first was the difficulty and the enormous labor involved in drawing conclusions out of extensive statistics. An immense amount of material seems to be there, and it requires a great deal of work to yield any definite conclusion. Some things strike me as of considerable value. One of these is that the statistics seem to bear out what others have observed—the general increase of blood pressure with age. If I remember rightly, nearly all his cases of 90 years of age and upwards had blood pressure of 200 or something like that, even though otherwise seeming to be in pretty good health. In that connection, I would be inclined to increase the limits he has taken for normal blood pressure at 130. At 60 years of age it seems to me that 140 or higher than that would be normal. Men who live to be 60 years of age differ from children and young adults, and if they have a normal, and we are to regard something as normal at that age, it is different from the normal of childhood and young life. I should say it would be 140 or 150. I have been taking blood pressures of elderly people for two years and a half and my experience is entirely in accord with that. Many of these were normal persons, except that they come for some error in refraction, and yet all showed rather high blood pressure. Another interesting observation was that those whose occupation had subjected them to hard labor seemed to show higher blood pressure than clerks and professional men and those who had not done hard manual labor. I think the difference reported is enough to constitute important testimony in this direction. I was much interested with reference to the influence of alcohol, which is apparently slight; and the much more positive influence of syphilis in these cases. But certainly age seemed to be the most positive cause of high blood pressure. I did not gather from these statistics that cataract was especially associated with high blood pressure, and this confirms what I have seen. The highest blood pressures have been in connection with albuminuria. In one of these cases there was incipient cataract. But I have seen a number of cases of mature cataract, and some of some years' standing, where the blood pressure was not high, for the patient's age. I would be inclined to expect more connection between the changes of blood pressure and degenerative changes in the chorioid. These seem more closely connected with vascular disease. It seems to me that cataract is not closely connected with vascular disease, but is most nearly explained by observations in the direction of a poisoning of the lens substance—some sort of autointoxication.

Dr. Lamb, Washington, D. C.:—In going over this work of arteriosclerosis for several years, and being much interested in it, I believe that the value of Dr. Greene's paper will come out later on when he follows up the information he has accumulated there by correlating it with other things, and follows up the patients themselves for some years to come. It is a big undertaking, but worth a good deal to the profession. The conclusions Dr. Jackson has drawn from the paper would accord with my own experience in studying these cases of arteriosclerosis in private practice and in clinic. In every case in the clinic where there are suspected retinal lesions, the blood pressure is taken. It is usually found high in those cases where the retina or chorioid, or a combination of the two are affected. So far as cataract goes, one might say generally it is more often associated with the lessened tension than with the increased tension, in my experience, though I have had cases of incipient cataract with arteriosclerosis, but they were not dependent one on the other. There are other conditions, in poisoning of the system through faulty metabolism, as Dr. Jackson has suggested, to indicate the same thing that causes the arteriosclerosis would cause the incipient cataract, regardless of the arteriosclerosis. I think we should thank Dr. Greene for so valuable a record, from which we can reason in connection with our work and get some valuable deductions.

Dr. Baker, Cleveland, Ohio:—I conceived the idea some years ago that there might be some connection between increased blood pressure and glaucoma. I have taken the blood pressure in all these cases I have had, and I can not say that I have been able to draw any conclusions from these examinations. The results have been so contradictory that it has been very unsatisfactory. I think part of my difficulty has been in the form of instrument I have been using. The instrument with the narrow band to go around the arm is unreliable and I think the one used by Dr. Greene is better.

Dr. Brown, Columbus, Ohio:—I want to say a word in regard to this paper because it was my privilege a number of years ago to spend a year at the Soldiers' Home as an assistant to Dr. Greene's staff, and more recently to witness a series of these examinations in which he was gathering data for this paper. Dr. Greene has succeeded in imbueing his staff with an active interest in this subject, and I can certify that the work has been carefully done, and believe that his statistics are in all respects dependable. The point has been raised in the discussion as to the association of the vascular changes and blood pressure with cataractous changes which were found, and it seems to me that possibly we may later have statistics which will add to the value of this contribution by selecting a series of cases in one of which lens and other eye changes are wanting and another in which the changes are more marked, and comparing the vascular finding in the two groups. It is quite common to find cataract with low blood pressure. Now just what are we to understand by this? The findings of Dr. Greene, which agree with those of observers generally, are that with age there is a gradual increase in blood pressure; though at a certain point, due to altered heart's action, this may again fall. But this fall is not indicative of absence of the pathological changes in the walls of the blood-vessels and those things which interfere with nutrition of the lens and other eye structures. We all know that lens opacities become more prevalent with advancing age. Can the statement, therefore, that we find cataract with low blood pressure be used to argue against the point raised in the statistics of Dr. Greene?

Dr. Fridenberg, New York:—The paper has interested me particularly as suggesting a number of other questions, some of which may be helpful. The first is as to the general question of standards and measurements of blood pressure as well as intraocular pressure; and, while the statisties are valuable, it seems to me the question of the standard has not been made as definite as it might have been. The authorities are at variance, and when we come to intraocular pressure there is still greater variance. It should be interesting to see whether the pathological rise of blood pressure in animals was accompanied by similar intraocular pressure. With regard to nicotine if you feed it to rabbits you will get increased blood pressure and changes in the vessels and heart. With regard to arteriosclerosis and what bearing it has on the ocular structures, there seems to be a tendency to make a diagnosis on ophthalmoscopic evidence of arteriosclerosis. When one vessel passes over or under the other it is supposed to give all sorts of symptoms. It would be interesting to know if there are any different eye symptoms where we have the crossing of a vein by an artery, whether that is within the physiological limit or whether it precedes the ocular disturbance in these cases. The statistics will be still more valuable when applied to concrete cases.

Dr. Young, Burlington, Iowa:—I think some of the confusion arising about the test for arteriosclerosis is because most of us are unfamiliar with it. I think the standard should be taken as we take it for vision. We arrive at the 20/xx by experimental process, and we arrive at the average blood pressure by the same process. Since reading an article on the question of arteriosclerosis, as a factor in the development of cataract, I have gotten a blood pressure machine, and I have done this for the simple purpose of determining whether there is any great deviation in these cases from what might be called an average. If we have a manifest deviation from the average it has a pathological significance. Blood pressure of 120, 130, 140, or 150, according to age, might not be considered a serious pathological process, but when above 160 or below 110, it has a pathological significance. I have been interested in a case lately, which I reported to the Chicago Ophthalmological Society about a year ago, a case of cerebellar tumor of tubercular origin. This case presented the typical appearance of Bright's retinitis. The urine examinations were negative, and finally the blood pressure was taken and shown to be only 70. On the other hand, recently a lady with slight fundus changes, claiming to be in the best of health, when I came to take her blood pressure, showed a pressure of 200, (at the age of 64). I think when we become more familiar with the use of the blood pressure machine we will have better appreciation of these things. I think with the gentlemen who recently wrote the paper in Dr. Alt's journal, that while it is not said that it has anything to do with the production of cataract, the presence of the high pressure in connection with cataract is a danger symptom, wherein we might expect postoperative difficulties, which is important for us to bear in mind.

Dr. Conner, Detroit, Michigan:—The subject that has been presented to us is certainly a large one and for myself I am very grateful for the observed facts. I could have wished, however, in connection with these observations on blood pressure, that there had been given to us the observations of the careful study of the urine, and alvine evacuations, etc., particularly as regards the lower bowels; in other words, the conditions of metabolism as associated with this blood pressure. That blood pressure of this high grade should have a relation to the formation of senile cataract is not improbable, but it does not seem to me that its direct influence could be great. I should expect more from its indirect influence; that is, its relation to poisonous materials and toxins which are so large a factor in the life of an old person. However, we want to observe every condition that is abnormal in a person in whom a cataract is being developed. If we knew all these conditions, then it would be fairly easy for us to eliminate some of them and so modify the development of the cataract and perhaps prevent its maturation. I hope the writer will use his large material to aid us in the further study of this matter.

Dr. Suker, Chicago:—We should not lose sight of the very important fact that arteriosclerosis does not always mean a generalized or disseminated arterial degenerative process, involving *pari passu* the larger and smaller vessels. Very frequently, in fact almost invariably, this connective tissue proliferation of the tunica intima and media first manifests itself in the so-called terminal vessels—coronary, cerebral, ocular, and the like—before the larger vessels become noticeably involved. For this reason we ought at all times make a careful inspection of the fundus and conjunctival vessels in any patient in whom we may suspicion vascular changes. Age in this particular is of no clinical significance. Arteriosclerosis can be quite pronounced in the very young; and this is not strange, when we come to realize that an infective process is the underlying cause for the vascular changes. Any infectious disease, either acute or chronic, will so leave its impress upon the vessel walls of young or old that given the proper impetus at some future time, angiosclerosis will eventuate. The question of high or low arterial blood pressure (tension) is practically only relative, just as Dr. Young has stated. You get increased arterial tension only when the larger vessels are fairly well involved. And what is more, this tension varies greatly during the 24 hours. An angiosclerosis more markedly and generally involving the terminal vessels will give an earlier rise in tension than when the process is more uniformly distributed throughout the larger and smaller vessels. There is not the slightest doubt but that the majority of our incipient senile cataracts are due to an angiosclerotic process, the same causes that bring about the angiosclerosis, involving particularly the long and short ciliary and chorioidal arteries and veins. And, by keeping these peripheral vessels well open by proper medication and causing an absorption of this connective tissue proliferation we can delay, if not entirely prohibit, the development of this type of cataract. The angiosclerosis is just as insidious and incipient as the cataract process. Stengel has well remarked that nothing yields more gratifying results than the early recognition of, and the proper treatment for incipient angiosclerosis. My clinical as well as private case records show quite a few instances of incipient senile cataract, dependent upon angiosclerosis, can be materially improved in visual acuity and the cataractous process practically checked. No doubt, Dr. Greene will find that many of his old soldiers are the victims of some toxic agent, either exogenous or endogenous, which is at the bottom of their angiosclerosis, this in turn accounting for the frequency of senile cataract among them. When once the angiosclerotic process is fairly well established little can be accomplished in either checking its onward progress or its consequences, among which latter is also classed incipient senile cataract.

Dr. Greene, Dayton, Ohio (closing discussion):—I have not kept a record of the number or proportion who are regular users of tobacco, but from long experience with the men I know that the proportion is large. I do not think Dr. Timberman quite understands the purpose of the paper, which is not to reach conclusions regarding the pathology or etiology of arteriosclerosis (or angiosclerosis, if you prefer that term), or its influence in the causation of certain intraocular diseases. I do not think we are yet in a position to reach conclusions; while we know something of the subject our deductions so far seem to have been theoretical rather than clinical. A broad and deep foundation based on further pathological investigation, clinical experience and statistical study must be laid; when this has been done legitimate conclusions can be reached. My purpose has been to contribute something to the statistical side of the subject. I have only departed from this general plan in quoting from Janeway ("Clinical Study of Pressure," page 118, et seq.) as to the relative effect of alcohol and tobacco on blood pressure. The question of the relation of arterial disease to age is a most interesting one, but it too must be more carefully studied before satisfactory conclusions can be reached. According to Metchnikoff ("The Nature of Man," page 228 et seq.) old age is practically a connective tissue period in the life of a predisposed individual during which the glandular elements of his organs and the coats of his blood vessels are overwhelmed by its proliferation and the same pathological processes take place in younger individuals under the influence of such poisons as syphilis, gout, lead and some forms of autointoxication. One of the most important questions is in relation to the association of arteriosclerosis or angiosclerosis, if any exists, to incipient cataract. Of 255 eyes with incipient cortical cataract only 5.9 per cent. had blood pressure under 130 m/Hg. Of 84 eyes with incipient nuclear cataract 26.2 per cent. had blood pressure between 130 and 160, averaging 139, and 73.8 per cent. had pressure of 160 and over with an average of 192.9. Of 102 eyes with incipient corticonuclear cataract 29.4 per cent. had pressure between 130 and 160, averaging 142, and 70.6 per cent. had pressure between 160 and 280 which averaged 179.8. Briefly summing up the statistics covering these three forms of cataracts, they show that of 441 of these 910 eyes showing some degree of lens opacity only 5.9 per cent. had blood pressure under 130. Dr. Jackson objects to this standard of 130 m/Hg for the upper normal limit, but one has to take some standard and there is no general agreement as to what it shall be (within certain limits). Janeway of New York takes 145 for the upper limit of normal blood pressure; Elliott of Chicago, takes 140. Considering the physical character of the men I have had to deal with I have taken the lower standard, but I have made it clear in the paper that I have taken this standard and have given my reasons for adopting it.

GLAUCOMA AFTER CATARACT EXTRACTION WITH IRIDECTOMY.

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Secondary glaucoma following cataract extraction with iridectomy is fortunately not very common. Its occurrence is generally said to be due to changes which obstruct the escape of fluid at the filtration angle. It is seldom possible to discover in the living eye what these changes are, but a microscopic examination of the excised eye usually shows some interference with the patency of the filtration angle. This obstruction may be due to some entanglement of the iris or capsule in the scar, or some obstruction of the pupil by membrane or inflammatory exudation. It is supposed to be more commonly due to adhesions of the pillars of the iris coloboma to the cicatrix and the lens capsule.

Treacher Collins[1] reports that in ten eyes blinded by glaucoma following cataract extraction, and microscopically examined, the filtration angle was closed in the neighborhood of the scar in every case. In one of the ten eyes the capsule was adherent to the scar, and in the tenth, from which the lens had been removed in its capsule, the hyaloid was adherent in like manner. In one case, in which a large iridectomy had been made and the anterior capsule partly removed with capsule forceps, the filtration angle was found blocked by an organized blood clot and inflammatory exudation and lens matter.

Priestley Smith[2] says that when an eye which presents no complication, such as entanglement of iris or capsule, which leads to traction on the ciliary processes, and which has enjoyed good sight for months after the operation and then becomes glaucomatous, we may reasonably conjecture that a transparent membrane, which at first gave no trouble, has lately contracted, or that it has become less permeable than before, and has been pushed forward by an accumulation of fluid in the vitreous chamber. He further states that the accumulation of fluid behind the lens in some cases seems to be due to obstruction of the circumlental space by swollen ciliary processes, and that changes in the hyaloid membrane, in the vitreous tissue, or in the fluid itself, are possible impediments to filtration, and in eyes blinded by glaucoma we often find the hyaloid and the septa of the vitreous thickened or coated by albuminous coagula.

The occurrence of glaucoma in an eye in which a broad peripheral iridectomy has been made, and in which there is no entanglement of capsule or pillars of the iris coloboma, would seem to support the theory of Bartels,[3] that the probable cause of glaucoma lies in a disturbed anterior ciliary venous circulation, and that it can occur in the absence of obstruction in the filtration angle. The following two cases have a bearing on this point:

Mr. B., aged 65, in good general physical condition, was operated for mature senile cataract May 13, 1905, a large peripheral iridectomy being performed at the time of the extraction. Recovery was uneventful, and six weeks after the operation there was a clear pupillary space, no evidence of iritic adhesion or entanglement of iris or capsule in the wound, and with correcting lenses the vision was 20/30. Four weeks later, during a severe attack of intestinal inflammation, he was attacked with glaucomatous inflammation. which terminated in complete loss of sight. Three months later the hardened eyeball was removed. On examination microscopically the operation cicatrix was found entirely free of iris or capsule entanglements and the filtration angle unobstructed. The ciliary processes were turgid and the chorioidal veins distended. It is thought that the inflamed ciliary processes poured into the aqueous chamber a serous fluid which escaped from the eye with greater difficulty than the normal secretion and clogged the filtration angle, thus producing the glaucomatous condition.

In the second case the conditions were quite similar. Mrs. C., aged 67, was operated for uncomplicated mature senile cataract Jan. 24, 1906, a large peripheral iridectomy being performed at the time of extraction. Recovery was uneventful, and four weeks later, without apparent cause, the patient suffered from severe pain and great impairment of vision. She was not seen until a week later, when the intraocular tension was found decidedly in-

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[2] Norris and Oliver's "System of Diseases of the Eye," 1898.

[3] Quoted from Tenny, "The Relation of the Circumlental Space to the Causation of Glaucoma," Trans. Sec. on Ophthal., A. M. A., 1907.

creased, the aqueous cloudy, but the anterior chamber only slightly shallowed. Vision had been reduced to light perception. Posterior sclerotomy was performed, as also paracentesis of the cornea, but with only temporary relief, and a few weeks later the hardened eyeball was excised. By microscopical examination no narrowing of the filtration angle could be discovered, though there was evidence of serous cyclitis which, by altering the constitution of the fluid in the anterior chamber, probably produced clogging of the drainage at the filtration angle. No entanglement of capsule or pillars of the iris coloboma in the wound could be detected.

These cases indicate that a broad peripheral iridectomy which frees the filtration angle is not always a preventive of glaucoma, even though iridectomy is recognized as one of the proper procedures to adopt in endeavors to cure the disease. They further indicate that a shallow anterior chamber and narrowing or closure of the filtration angle are not necessary for the production of glaucoma, but that alteration in the character of the intraocular fluids may interfere with the proper drainage from the eye, resulting in a retention of fluid and increased tension. Quoting from Priestley Smith,[4] "The evidence is complete that retention is the essential factor in the morbid process."

It follows, then, that secondary glaucoma following cataract extraction with iridectomy is not absolutely avoidable by any particular method of operating, though glaucoma is less likely to occur following a clean peripheral iridectomy in cataract extraction that heals without incarceration of capsule or iris in the wound, or morbid changes which narrow or close the filtration angle.

Glaucoma following discission for secondary cataract is not infrequent and is due to not only the pressure upon the filtration angle, resulting from the swollen ciliary processes, produced by traction, but also to the altered character of the fluids of the eye, caused by the exudates thrown out by the inflamed ciliary region.

The treatment, as in any other morbid condition, resolves itself into attempts to remove the causes. The exciting causes are the various conditions which disturb the circulation and congest the ocular venous system. Aside from the effects of trauma accompanying the operative procedures, we may have exposure to cold, depressing emotions, marked nutritional disturbances, or general debility of any character as causative factors. Warmth, rest, elimination and tonics are, therefore, essentials in proper treatment. In my judgment, a rheumatic tendency is one of the most active predisposing influences, and accordingly the administration of sodium salicylate in fairly large doses will prove beneficial, especially when given in connection with calomel purgation. Pilocarpin by hypodermic injection will also be found beneficial in those cases where the drug is well borne. Pilocarpin locally is indicated in those cases where there is a tendency to blocking of the filtration angle mechanically by folds of iris.

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[4] Erasmus Wilson Lectures on the Pathology and Treatment of Glaucoma, 1891.

As surgical treatment the procedures to be adopted should depend upon the conditions presented. With glaucomatous symptoms in an eye with reasonably deep anterior chamber and no determinable evidence of blocking of the filtration angle by iritic or capsular adhesions, or inflammatory exudate, thus pointing to an alteration in the constitution of the fluid rather than a mechanical obstruction of the opening in the filtration angle, paracentesis of the cornea, repeated if necessary, will usually prove sufficient when carried out in connection with the other general treatment indicated. If the anterior chamber is shallow and there are other evidences of narrowing or closure of the filtration angle, an anterior sclerotomy is preferable, and with a view to still further lessening the intraocular tension this may be supplemented by a posterior sclerotomy. If the anterior sclerotomy wound can be kept open, under as nearly aseptic conditions as possible, or reopened in the event the intraocular tension remains high, while the patient is at the same time receiving appropriate treatment for the disturbed ocular circulation, the ultimate results will be more favorable.

This latter point was impressed upon me by a unique case operated for senile cataract some ten or twelve years ago. The patient, a distinctly rheumatic person, apparently made an uneventful recovery from an extraction with iridectomy until the tenth day, when he had a sharp attack of articular rheumatism accompanied by increased intraocular tension, shallowing and clouding of the anterior chamber, ciliary injection, and pain in and about the eye. Soon after the onset of this attack the patient by accident struck the eye with sufficient force to partially rupture the wound and permit escape of aqueous. There was almost immediate relief from pain, and in a few days the glaucomatous symptoms had practically disappeared, but the fistulous opening at the limbus did not close entirely for several weeks, and only then under the effects of stimulation, which produced a permeable cystoid scar, one of the surest and safest preventives of secondary glaucoma following cataract extraction. It is thought that the patient had an attack of cyclitis with the exudation of serous fluid of such consistency that it did not drain well through the filtration angle, producing glaucomatous symptoms from which he was relieved by large doses of sodium salicylate coupled with the traumatic re-establishment of drainage. Good vision with the correcting lenses was retained for a period of three years, or until his death.

Priestley Smith,[5] referring to primary glaucoma, says that a certain group of cases depend on disease of the blood or blood vessels, causing an excess of fluid in the vitreous chamber, and that the swollen ciliary processes prevent exit of this fluid. He further says that exudations into either the aqueous or vitreous chambers are sometimes followed by high tension, and whether a cause or a consequence of the glaucomatous complication it aggravates and perpetuates the difficulty of filtration.

There is reason for believing that alteration in the constitution of the intraocular fluids may occur in secondary or any other form of glaucoma, and that failure in drainage is as often due to the altered character of the fluids of the eye as to mechanical obstruction, and that this alteration, in any form of glaucoma, depends primarily on some vascular disturbance which congests the uveal tract.

I am a firm believer in the retention theory as to the cause of glaucoma, and have not been able to convince myself that a mere obstruction of the filtration angle at one point in the filtration zone, as occurs when a tag of iris or capsule is incarcerated in the wound, can in itself account for the increased intraocular pressure. Every operator of experience has seen numerous cases of incarcerated iris or capsule which did not produce glaucoma, and there is no logical reason for believing that such a condition, from mechanical obstruction of a portion of the filtration angle only, can alone produce glaucoma. If it is a factor in the production of glaucoma it is more reasonable to assume that it excites an inflammatory change in the uveal tract which results in a constituent change in the fluids of the eye, impeding proper filtration.

Evidence seems to show that glaucoma results from excessive pressure in the vitreous chamber, caused, as pointed out by Tenny,[6] by diseased blood vessels in the uveal tract. At the same time there is probably an alteration in the constitution of the aqueous humor, which blocks up the outlets. This being true, our treatment should not only be surgical to relieve the eyeball of intraocular pressure and do away with purely mechanical obstruction to free drainage, but constitutional also for the purpose of favorably altering the character of the fluids of the eye. This treatment will essentially be that treatment which relieves congestion and gives appropriate attention to recognized constitutional dyscrasias.

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[5] Norris and Oliver's "System of Diseases of the Eye," 1898.
[6] Trans. Sec. on Ophthal. A. M. A., 1907.

DISCUSSION.

Dr. Jackson, Denver, Colo.:—This is an extremely interesting paper and brings up certain points that have recently been advanced and perhaps established. The investigations of Uribe-Troncoso have shown the alteration of the aqueous in the case of glaucoma as well as cataract. Raehlmann, with the ultra microscope found that in the aqueous after discission of the lens and also after it becomes more albuminous, are found minute particles which tend to accumulate in the tissue of the pectinate ligament and to clog its spaces. Glaucoma has been produced experimentally by metallic oxids accumulated in those spaces. Now with reference to repeated paracentesis, it has been shown that it alters the composition of the intraocular fluid, rendering it more albuminous. I should be afraid to do it in cases where the angle of the anterior chamber was not closed, but was blocked by accumulations. It seems to me in these cases the rational procedure is to get a fistulous scar, and permanent opening from the anterior chamber under the conjunctiva. The operation recommended by one of the Indian surgeons for this purpose I have recently tried. It is not difficult of execution and worked very well in a case of secondary glaucoma. In making the incision for iridectomy he uses a very narrow knife. Before the edge of the knife emerges from the sclera, and after it has entered the sclera entirely, he turns it forward and makes a cut partially through, from the inside. He then turns the knife back and cuts out. This leaves a triangular piece of tissue, almost cut off except the superficial edge towards the cornea. The nutrition of this piece is greatly interfered with, and the result is a scar that remains permeable to the fluids. He was led to this by several accidental cases where it occurred. I recall a similar case where the tissue was separated, by making too small an incision leaving a long slip of tissue attached at one end that stood in the way of healing. It seems to me that something of that kind that will give a permanent outlet is the solution of the treatment of these cases.

Dr. Minney, Topeka, Kansas:—Some eight years ago I had to remove an eye because of sympathetic trouble. Glaucoma set up in the other eye. I did a broad iridectomy, which relieved it. I did a paracentesis on that eye some forty times and I was two years in treating the case. That was eight years ago and the eye is well to-day. I think a frequent paracentesis in these cases is worth trying. The one case I know of was successful. I do not know whether the angle was blocked or not, but such was the result. I think this theory was all right in its place, but the practical application is what we want, and this case was certainly saved and the eye has perfect vision to-day.

Dr. Fridenberg, New York:—I would like to call attention to some of the practical sides of this paper which appeal to me very much. We all recognize that neurotic conditions and the peculiar conditions of the blood have their effect, but we should bear in mind that glaucoma developing after operation is almost invariably due to mechanical conditions. It seems to imply faulty technic. With perfect technic we would not have it. That brings us to the question of prophylaxis and treatment. I think certain steps in the cataract operation are partly to blame for its development. It was to avoid the traction and tension that peripheral capsulotomy was devised. We may often cut short an acute attack of glaucoma by a free discission, and the after-operation for cataract is really a prophylactic operation when performed in the right way. The capsule is the dangerous thing. If we had a method of opening which would keep the tags away from the section, we would have comparatively few cases of secondary glaucoma. From this point of view the simple operation is satisfactory, and there is no doubt an uninjured iris keeps it away better than when we have a coloboma which may allow the capsule to prolapse into the wound. The ideal operation from that point of view is the removal of the lens in the capsule, no matter what the other disadvantages of this procedure. The question of the treatment of an attack of acute glaucoma is an important one, and it seems to me that the operation Dr. Jackson has spoken of, the formation of a fistulous scar, is to be striven for. A simple method is to open back of the original section and form a conjunctival flap by simply paring the anterior scleral lip with the scissors, and you have a section covered by a conjunctival flap which tends to remain patulous. The choice of the cataract extraction method is of great importance with reference to postoperative glaucoma.

Dr. Young, Burlington, Iowa:—I have had three cases of postoperative glaucoma, two I know, and I think a third, not recognized at the time, which was early in my practice. The first was a woman 80 years of age on whom I made a simple extraction. She had had an unsuccessful extraction in the other eye by a colleague in another city. As this was from irido-chorioiditis I thought she had a tendency to iritic trouble. Before closing the eye, I put in a drop of 1 per cent. atropin. She had a full fledged attack of glaucoma 12 hours later. The second was the case of a young woman with rudimentary irides. I made the same operation on her, of course, the simple extraction. Twelve hours later she had an attack of acute glaucoma. Like the former case, she had eserin for five or six hours, and made a good and permanent recovery. I believe, as Dr. Fridenberg has said, incarceration of the capsule is by all odds the greatest cause but I can not understand why rudimentary irides should, as some writers maintain, predispose to postoperative glaucoma.

Dr. Bulson, Ft. Wayne, Ind. (closing discussion):—I thought I made it plain that I was discussing that class of cases in which the filtration angle is not mechanically blocked by iris or capsule. However, I fail to understand how a small tag of capsule or iris in a very limited portion of the filtration zone precipitates an attack of glaucoma. I believe that other factors are responsible for the glaucoma and that blocking of the filtration angle by tags of iris or capsule is but an incident and while perhaps a contributory cause is so by exciting disturbance in the uveal tract. That the glaucoma may occur entirely independent of any such conditions is certain, as the cases reported indicate. Repeated paracentesis does relieve these cases; and, in the meantime if by suitable treatment the character of the intraocular fluids can be favorably altered the glaucoma is cured. A cystoid scar, so common in many cases following cataract extraction, is a beneficial result, and it undoubtedly prevents what would otherwise be a glaucomatous condition, and its good effect is due entirely to the fact that it brings about a constant leakage of aqueous which prevents injurious intraocular tension. I believe that the tendency to glaucoma following discission operations is due entirely to the traction on the ciliary body with its attending increased congestion of the uveal tract and possible alteration in the character of the intraocular fluids. Not every uveal congestion or inflammation is attended by increased intraocular tension, but it seems quite rational for us to believe that there is a direct connection between certain forms of uveal inflammation and glaucoma, and that the increased intraocular tension is due to retention as a direct result of the altered constituency of the fluids. There is room for further study along this line, but as therapeutic measures we are safe in adopting such as are known to reduce tension and relieve the inflammation upon which the altered character of the fluids depends. The increased tension is relieved by paracentesis, and sclerotomy, either anterior or posterior, or both. The uveal congestion or inflammation is generally favorably affected by suitable eliminants, the administration of large doses of sodium salicylate, and the local application of heat.

SOME EXPERIENCE WITH SIMPLE GLAUCOMA AND CONCLUSIONS THEREFROM ON THE RELATIVE VALUE OF OPERATIVE AND NON-OPERATIVE TREATMENT.

H. B. Young, A.M., M.D.

BURLINGTON, IOWA.

Whether the operative treatment of non-inflammatory, or simple glaucoma, is to be considered the *sine qua non* that it is in the inflammatory type is, I take it, a question that will not fail of discussion so long as three questions precedent remain unanswered. These three questions are:

1. What is the pathology of glaucoma?
2. How shall we distinguish non-inflammatory from inflammatory glaucoma?
3. What is the measure of relief attainable in this disease by either mode of treatment?

On the first of these I have nothing to offer. On the other two I wish briefly to present some views acquired by experience and study of the reports made by others. I do not pretend to say that my experience has been unusual or extensive—just the average, I fancy, of the environment—but it has been sufficient to bring me into practical touch with all the perplexities of the situation, and not the least of these has been the realization that my patients sometimes go, or have gone, to colleagues who entertain classification and therapeutic beliefs not in harmony with my own—a most prolific source, as all must admit, of mutual embarrassment and probable harm to the patient. It is because of this more than anything else that I am led to again ask your consideration of this subject.

HOW SHALL WE DISTINGUISH NON-INFLAMMATORY OR SIMPLE GLAUCOMA FROM INFLAMMATORY GLAUCOMA?

A review of the literature, particularly that of the text-books, will very quickly uncover the fact that with most writers chronic glaucoma and simple glaucoma stand alike for non-inflammatory glaucoma—are interchangeable, if not, indeed, synonymous terms. The difficulty, in some cases, of differential diagnosis between simple glaucoma and optic nerve atrophy is always mentioned; likewise the slowly increasing dimness of vision without other disturbing symptoms. Tension may be increased moderately or not at all, and the pupil may or may not be dilated and sluggish. In short, it is atypical glaucoma as compared with inflammatory or congestive glaucoma, "inflammatory or congestive" relating to the great or moderate injection of the scleral vessels.

Under such description the following case, of which you have all seen the duplicate, could be classed as non-inflammatory glaucoma:

Mrs. N. M., aged 62, first seen May, 1906, comes for cataract operation, which an "eye specialist" said would restore her sight when blindness was complete; L. E. weak for years, very little vision the past two years. R. E. began to fail four months or more ago; never any pain or inflammation in either; vision just smoky, and the lamp sometimes has colors around it.

St. pr.; the L. E. has T. + 2 and p. l. in a very small area of the extreme temple field. The cornea is bright. The pupil measures 4 mm., is sluggish, and the anterior chamber very shallow. The lens is too opaque for a view of the fundus.

The R. E. has T. + and 20/50 V., with 2 D. H. corrected. The F. V. can not be accurately mapped because of the patient's extreme nervousness, but shows no gross defect. The pupil measures 4 mm., is sluggish and the anterior chamber shallow. The nerve is dusky, with uneven margins, but not characteristically cupped.

I saw this patient at regular intervals until November, 1906. During all these months the systematic instillation of pilocarpin, plus a general hygienic regimen, held the conditions in *statu quo* and gave better use of the eye. To the patient my tentative treatment seemed all sufficient and my talk of operation a needless harm.

But judged by the standard set up years ago by Schweigger, that "a dilated sluggish pupil and shallow anterior chamber precluded the diagnosis of simple glaucoma, meant, on the contrary, inflammatory glaucoma, dormant perhaps, but still inflammatory glaucoma," or Abadie's declaration, that simple glaucoma was glaucoma of the posterior segment of the globe, while glaucoma of the anterior segment was inflammatory glaucoma, this case was really one of inflammatory glaucoma, and such it proved to be.

About December 1 she got infective "sore throat" epidemic in that locality. There was bowel derangement and fever for two days; then the R. E. became inflamed and painful and V. sank to qual. p. l. When I saw her, three days later, the crisis had passed, but there was T. + 2, the cornea was steamy and V. only 10/150. Iridectomy (and it was most difficult to accomplish) fortunately reduced T. to normal and restored the 20/50 V., which is still maintained, although there is bitter complaint of light reflections and there is now only 1 D. H. The L. E. has remained quiet, but 1 p. has been lost.

Cases of inflammatory glaucoma which have very short prodromals and about which there is no question of diagnosis are scarcely more common than this variety, but in the few I have seen with normal pupils (and I wonder if anybody has seen many such) I have never seen inflammation and rapid loss of vision, and one which I will presently report I have had under observation for twelve years. Schweigger's distinction, therefore, although it seems to have received little notice, appeals to me as logical in every way. With such a distinction we could be rid of the confusion arising from the indefinite use of the word "chronic." It is superfluous in non-inflammatory or simple glaucoma, which is always chronic or slow, and such cases as the one reported could have it properly attached as indicating that form of inflammatory glaucoma. If we could have an agreement upon this as we have upon the prism measurement of muscular dynamics, it would, regardless of what pathology shall eventually tell us, be a step toward a ground of understanding among ourselves which we seem not now to possess.

WHAT IS THE MEASURE OF RELIEF ATTAINABLE, ETC.?

The most recent systematic utterances in this connection are probably those of Posey and Risley before the Ophthalmic Section of the American Medical Association in 1906 and 1907, respectively.

The measure of relief by non-operative means seems to be fairly stated by Posey. The disease was held largely in abeyance in a considerable number of cases for long periods of years by a certain use of miotics.

The measure of relief by operative means was represented by Risley (who cited 7 cases with 7 eyes saved out of 12 operated) as curative, and as to numbers, provided it was done early, largely in excess of previous estimates; notably those of Berry, a strenuous advocate of operation, who accepts Horner's statistics (1882) showing less than 1/3 as many improved and three times the loss when compared with the same in inflammatory glaucoma.

That the evidence submitted failed of being wholly conclusive is manifest from the remarks of those who took part in the discussions; but to my mind the feeling of uncertainty which prevailed might have been dissipated if the point had been raised that part of the evidence was not germain to the issue. Suppose, for example, that I had operated before the inflammatory outbreak in the case I have reported, or in another just like it that I operated twelve years ago, seen recently and retaining the restored 20/30 V., without farther limitation of the field and the blind eye never having given any trouble!

To include these, as might in that event have been done, in the list of cured non-inflammatory cases would certainly be misleading, and yet cases not so unlike these were here quoted. In the 14 eyes involved the dilated pupil and shallow anterior chamber was recorded in 7 (not described in 7); the steamy cornea in 2; tortuous scleral vessels in 1, and pain in 9. These conditions were variously distributed, but in the 14 only two escaped this record entirely. The 60 per cent. relieved is also so near the record for inflammatory glaucoma that confirmation may be had of previous doubts.

However, in Hasket Derby's case, reported by Cheney, we find a better compliance with the Schweigger standard, and it is interesting to note that in this case, listed as "improved," the 5/24 V. "fell off immediately after operation, to recover only slowly and partially"; 3/25 after two years; the same after twelve years; then a slow retrograde but something left after twenty-four years. To compare with this I will report the case I have referred to:

Mrs. M. M., aged 60; first seen May, 1895. She discovered eleven years ago a "V-shaped" blind spot in and down in the L. E. This gradually spread until she had only qual. p. l. up and out in that eye. It has been better rather than worse the past year. But during this time a cloud has come over the R. E. There has never been any pain or inflammation in either. She wears + 3 sph. for distance, + 6 sph. for near.

St. pr. (1895): V. O. D. is 20/70 + and O. S. fingers at one foot up and out. Tension is normal (?) in both, scleræ white, corneæ bright, pupils 3 mm., anterior chamber normal (?), the left disc dusky and fairly cupped, the right cupped but not so markedly, and the nasal field limited 20° in the median line (more below). She is otherwise in good condition, mentally as well as physically, anxious, of course, but calm.

After canvassing with her the prospects by the two methods of treatment, pilocarpin was ordered, although I regret to say not so actively as Posey advises. Three years later (1898) the conditions were: V. O. D. 20/100, the nasal field a little more defective and the nerve more plainly cupped, O. S. unchanged. About this time she visited in New York and consulted Dr. Knapp, who confirmed my diagnosis of simple glaucoma, advised more pilocarpin and watchfulness for the possible need of operation. Five years later (1903) V. O. D. was 10/200 with well-marked cup, and O. S. qual. p. l. with deep blue-green cup. When I again spoke of operating she said she could still with a loup see the writing on her business papers when held close, and, as the rate of decline was slow, she figured that the chance for useful vision for her life expectation was too great to take even a moderate risk by operation. Two years later (1905) I was called in haste to see her, the message being "a sudden great loss of vision." I found her with qual. p. l. only, but no apparent change in the conditions otherwise. The breath had a foul odor and there was loss of appetite with constipation; so it occurred to me that there might be coincident toxic amblyopia of intestinal origin. Calomel and salines were ordered, and the former vision was promptly restored.

St. pr. (Aug. 31, 1907): Central vision is practically abolished and she has annoying subjective images, either landscapes without the green of the foliage or cloth designs in which red and green predominate. Temporal vision in the right still enables her to get about quite well, and the left still has qual. p. l. Tension and pupils are unchanged. She has for the past year neglected the pilocarpin, but will resume it.

What her vision might have been now if she had persisted with the pilocarpin is, of course, pure speculation (ditto by operation), but it seems not too much to assume that with useful vision after twelve years of indifferent treatment a better standard might have been maintained by consistent medication; nor is it too much to assume that so long as we can see cases like this—that go twenty-three years without total blindness or disturbance of the anterior segment—one may properly doubt the possibility of change to the other category which some claim is the end of all glaucomas. Moreover, the argument devised to show that tension is always the underlying cause of nerve destruction is capable of different interpretation, for a tension that is doubtful to the educated touch, but still too great for the nerve to safely bear, should first indicate a nerve too far from normal to bear ordinary pressure; in other words, a posterior disease process, and until we know glaucoma to be a disease entity of the globe rather than a "syndrome," as Wilder suggests, a glaucoma posterior.

Taking, then, the result in Derby's case as typical of the measure of relief by the operative method, a fair estimate because it is in harmony with the earlier experience, and the result in Posey's case, supplemented by my own as typical of the same by the non-operative method, it would seem that the prospects are more for palliation than for cure either way, and the relative value of the two methods becomes a matter of computation between the *risks* of sudden total loss, postoperative cataract (one of my patients got this, and extraction was subsequently a failure through iridocyclitis) or "dazzlings," and *chances,* remote, for permanent relief and, average, for a longer lease on vision. The balance may be in favor of the operative method, but it is surely not great enough to warrant criticism of the colleague who says to his patient: "Take your choice."

Per contra, although it is not strictly within the purview of the title, the balance is overwhelmingly in favor of the operative method in those cases showing either the dilated sluggish pupil with shallow anterior chamber, steamy cornea, pain, or rapid loss of vision, no matter how free from inflammation they may seem to be. But these under the premises should not be mistaken for non-inflammatory or simple glaucoma.

DISCUSSION.

Dr. Lewis, Buffalo, N. Y.:—Dr. Young has distinctly added to our knowledge by his discrimination between chronic and simple glaucoma, and I am very glad to have heard the conclusions at which he arrives. I realize more and more that in the eye are manifested many constitutional disturbances, that originate in other organs. I believe as we are growing better physicians we are growing to be better ophthalmologists, and one of the most promising signs of the times was evidenced yesterday when we, as ophthalmologists, were trying to determine what bearing blood pressure had on ocular conditions. In the concluding case Dr. Young cited, I think it is evident that toxemic conditions antedated the blindness for some time. As we get better acquainted with the various poisons and their different manifestations, we may find that this form of simple glaucoma is really a toxemia, and when we have called to our assistance the aid of the generalists and have found what various blood tests and other methods of accurate diagnosis will determine, we may learn that this complex of symptoms is not really glaucoma at all. In calling attention to a reform of the advisability of surgical intervention in these conditions, Dr. Young has done well, and I know of no work which would better repay careful general research than that of the general toxic conditions in their relation to what we term simple glaucoma.

Dr. Jackson, Denver, Colo.:—The bringing before us and the keeping before us of the discrimination as to the different kinds of glaucoma is needed, to clear up our ideas as to its therapeutics. At the meeting of the Ophthalmological Society at Washington, Dr. Bull read a paper on this subject, and Dr. Risley raised the question of what was simple glaucoma, and criticized Dr. Bull's classification of cases. Dr. Young has, I think, with clear reason, criticized Dr. Risley's paper of a few weeks later. We ought to be past the stage where all glaucomas are regarded as one pathological condition. I agree fully with Dr. Young's discrimination. That other cases are confused with simple glaucoma is responsible, I think, for some of the good results of operation in simple glaucoma; cases that are entirely free, or have been up to the time of operation, and afterwards remain free from inflammatory attack, but have well-marked exacerbations. They have attacks with somewhat dilated pupil and impaired vision and often decided rise of tension. The point has been made that the rise of tension is more apt to occur late at night than at any other time in the 24 hours, and it is worth while to visit the patient at such time and test the tension. These cases, otherwise simple, that have gone on with slow impairment of vision, but with exacerbations have yielded to operation as well as cases of inflammatory glaucoma. I would rather divide glaucoma by the line of exacerbation, although I think that even simple glaucoma at a late stage predisposes the eye to inflammatory attacks which simulate inflammatory glaucoma.

Dr. Green, Jr., St. Louis, Mo.:—I was especially interested in Dr. Lewis' remarks with reference to a search for some underlying constitutional cause in cases of glaucoma simplex. Several years ago a young woman came to me with typical glaucoma simplex, the disease having progressed in one eye to total blindness. In the other vision was 16/xix, with some narrowing of the field. Both nerve heads were deeply cupped. I managed the case for several months with eserin and pilocarpin, but vision failed to 16/lxviii. Reviewing the general medical history of the patient I found that she had suffered since early childhood with intractable constipation. An internist who saw the patient in consultation discovered a congenital deficiency in the muscular tone of the stomach and intestines. I believe that the continual resorption of noxious material from the intestines had given rise to an optic neuritis and that subsequently, through some inherent weakness of the lamina cribrosa, the disc had become cupped. Concomitantly with the institution of treatment appropriate to the intestinal condition, vision improved to 16/xv and has remained so up to the present time. In addition to the search for and correction of any constitutional error, the local treatment with miotics is of the first importance. I have gained the impression that the majority of ophthalmologists are inclined to place little reliance on miotics. Possibly this distrust has arisen through the fear that the patient may not consistently carry out the treatment. To gain the best results the patient must be made to understand the gravity of his condition and necessity of the strictest adherence to the course laid out by the physician. Failure to secure intelligent and active coöperation on the part of the patient probably explains some of the failures incorrectly ascribed to the use of miotics.

Dr. Alt, St. Louis, Mo.:—While I agree with almost everything Dr. Young has said, I want to say a word with regard to what Dr. Green has said in reference to congenital weakness of the intestinal organs causing constipation and a toxemia which might produce simple glaucoma, that is, that if as common an ailment as constipation and autointoxication can cause simple glaucoma, it is astonishing, it seems to me, that we do not see a great many more of its kind. Another question, however, seems to me to be of more importance, namely, whether a simple glaucoma or posterior glaucoma may not have some connection with affections in the accessory sinuses. In this connection I would recommend all of you to read the recent monograph by Onodi on the optic nerve and its relation to these sinuses which is magnificently illustrated by photographs in natural size and shows how easily the optic nerve may be directly influenced by diseases of the accessory sinuses.

Dr Lamb, Washington D. C:—There are just three points I would speak of: First, during the periods of remission, between attacks of hypertension with dimness of vision, we may get even a minus tension in simple glaucoma. Second, operation should be done when the field is only slightly contracted and the vision only slightly impaired, to be successful for any length of time. Third, and one that is essential to know and perhaps the hardest to decide, is that your patient must stimulate you in some way to an understanding as to which method is better for that particular case, as to whether they will cooperate with you or not, or whether the operative interference ought to be made at once; I mean cooperate with you in the pilocarpin and eserin treatment.

Dr. Young, Burlington, Iowa (closing):—In my paper I have undertaken to give both an argument and a protest: an argument for a more exact classification of glaucoma clinically, so as to avoid the confusion which has arisen, and a protest against the tendency to dogmatic insistence on operative treatment for conditions not fully understood. If the argument is good the protest follows as a natural sequence. I hope that this organized body of ophthalmologists will take the matter up on the lines I have suggested. I realize that it is not exactly in good taste to make much protest about the actions of those in position to speak more authoritatively than I can, but I have it in mind to insist that we see glaucoma in at least three forms. First we have one that we all agree must be operated on. The second is that which follows injury or operation for cataract, in which there is frequent and permanent relief with miotics. Then we have a third on which perhaps the majority would operate (simple glaucoma). But that majority is not united on the method by which this should be done. One says, "a mm. section," another, "1/3 of the iris." A great deal has been accomplished by improved technic and asepsis, but I do not believe that these can be credited for the amount claimed. The older men in ophthalmology cannot be charged, in my estimation, with a lack of critical observation. They operated successfully for inflammatory glaucoma, possibly not so well as we do; but I think that something should be allowed for their judgment in that they were not so successful with simple glaucoma. Nor does improved technic always make for success in other ways. The best result I ever got from iridectomy was from the poorest I ever made; while one of the best resulted in total failure. It thus comes down at last to where one must ask, as I do in all cases: If I had this condition, what would I have done? That removes it from the field of academic discussion and makes it pre-eminently, as it should be, one of personal application. And if I am satisfied—if the majority can be shown to be benefited by operation—I do not care anything about the risks. I have led "forlorn hopes" in the past and may perhaps undertake others; but I want to know that the evidence is exactly to the point so that I may have something definite to refer to when I get a failure.

ANGIOSCLEROSIS OF THE EYE.

ROBERT SCOTT LAMB, M.D.
WASHINGTON, D. C.

The effort to find a subject of interest and of vital moment to one's collaborators is not easy, and I have selected arteriosclerosis or angiosclerosis and its effects on the structure of the eye, in the hope of bringing to you something for consideration that has been of much interest to me and benefit to my patients, and may perhaps be not without profit to us all.

It will not be amiss to review in brief the composition of the normal structures that we are considering, after naming the causes that lead to the pathologic condition; we may the better appreciate the changes that take place and their relative effects and understand why certain symptoms occur, subjective and objective.

The causes of angiosclerosis may be summed up as follows: Congenital anomalies, syphilis, faulty metabolism, toxic agents, including alcohol, tobacco, and the toxins of the organisms of acute infectious diseases.

To be brief, the eye is formed chiefly from the invaginated ectoderm and the enclosed mesoderm that attains its position through the chorioidal fissure and becomes so important a factor in maintaining ocular nourishment during the entire existence of the eye. From the mesoderm are formed those parts with which we will concern ourselves, and in them we find vascular tissue, namely, the vessels of the nerve fiber layer of the retina; the chorioid, iris and sclerotic are the most extensive outgrowths of the mesoderm.

We will consider these parts in the order named. The nerve fiber layer of the retina contains all the larger vessels of the retina. The arteries have an intima lined with endothelium, muscle fibers and an adventitia rich in elastic fibers, the outer of which run longitudinally.

As the vessels diminish in size, the media is the first tunic to disappear. The veins, lined also with endothelium, have almost no media, but are very rich in elastic fibers. The capillaries are mere endothelial tubes. There are no lymph vessels, but there are lymph spaces around the blood vessels, the so-called perivascular lymph spaces. It would be well here to state that these blood vessels lie within the nerve fiber layer, between the internal limiting membrane and the ganglion cell layer, in contact with the non-medullated axis cylinder processes of the ganglion cells, and this proximity has much to do with the pathology of these structures.

Normally the chorioid is composed of the membrane of Bruch, a homogeneous structure lying between the retina and the choriocapillaris and joined to this layer by some elastic tissue. The choriocapillaris is a network of fine meshed capillaries, becoming coarser or more open toward the ora serrata. The layer of medium-sized vessels contains much fine elastic tissue with a few pigment cells and endothelium separating it from the choriocapillaris on the inside and the larger vessels on the outside. Its veins are without muscular tissue, and their endothelial lining is separated from the adventitia by the perivascular lymph spaces of the choriocapillaris.

The arteries are composed of an endothelium, a media with circular muscle fibers and an adventitia with longitudinal muscle fibers, together with perivascular lymph spaces. The layer of large vessels, chiefly veins, is imbedded in membrane of pigmented elastic fibers.

The suprachorioid is a pigmented elastic-fiber membrane, with endothelium between it and the lamina fusca of the sclerotic, assisting in the formation of the suprachorioidal lymph space and communicating with the capsule of Tenon.

The sclerotic is pigmented and is united to the chorioid by pigmented connective tissue, lined with endothelium to form the part of the suprachorioidal space. This endothelium invaginates all the muscles, nerves and vessels crossing this space. The sclerotic is composed of white and elastic fibers, distributed in bundles, running longitudinally, circularly and diagonally, one or another arrangement preponderating in the different parts of the globe, depending for direction on the necessary function. It contains a few vessels and contains the origin of the posterior ciliary veins and, in part, the anterior. Its arteries are derived from the shorter posterior and long or anterior, and the ciliary branches of the posterior, forming the anastomotic ring around the optic nerve by uniting with the branches of the central retinal veins.

The early pathologic changes in the vessels are, of course, similar in all tissues, differing only in so far as surrounding tissues permit. With the slightest disorganization of the vessel wall there is a transudation from the vessel into the contiguous tissue. In the retina this causes an edema that may be limited to the nerve fiber layer, and is readily remedied, but, on the other hand, may affect the outer nuclear layer, in which case complete recovery cannot take place. If the inflammation progresses beyond the edematous stage an exudation of plasma and polynuclear leucocytes occurs into the adventitia and perivascular space, and we have the beginning silver wire vessels. In the smaller vessels the endothelium proliferates and there is increased connective tissue formation.

These changes usually occur first in the retina, where the vessels are smaller; next the inflammatory process appears, in the choriocapillaris; next in the medium-sized vessel layer; next in Haller's layer, and later still degenerative and proliferative changes are found in the sclera.

The symptoms noticeable to the patient are muscle tire, with headache attendant on the use of the muscles; scintillation scotoma; slight or prolonged blindness, or blurring vision, and this is sometimes the only symptom from which the patient feels discomfort, and is the reason that many persons are seen in the early stages who are seeking relief from this single disturbing feature. The objective symptoms are a conjunctivitis, a partial ptosis, nystagmus, blepharospasm, possibly a marked pulsation of the temporal arteries and carotids, and, with a sphygmomanometer, an high arterial tension (which, though usual, is not always present). With the ophthalmoscope a hazy disc and retina, possibly obscuration of the capillary vessels and apparently nodes or interruptions in them as they cross the disc or approach the macular region, with a tortuosity; white lines or bands of the larger vessels; arterial pulsation, and perhaps spasm; aneurismal dilatation; perhaps hemorrhage into the retina and chorioid, or the chorioidal vessels may show white streaks or bands; chorio-retinitis or neuro-retinitis; or any of the ophthalmoscopic signs of degeneration or proliferative changes. The fields are liable to be contracted in proportion to the amount of destruction present.

The diagnosis is easy or not, according as one by experience is familiar or not with slight changes in the color and shape of the retinal vessels; I say slight because, of course, the gross lesions are very plain, but the disease has progressed at this stage so far that it is less amenable to treatment.

The earliest ophthalmoscopic signs are the slight haze and indefiniteness of the disc and surrounding retina, with possibly obscuration of the capillary vessels, apparent nodes or interruptions in them; together with a tortuosity of the capillaries about the macula or at the periphery; and later, when the round cells enter the adventitia and perivascular lymph spaces, the beginning white lines along the vessels that later become broader and the vessels appear as white bands. As the vessels become disintegrated and connective tissue forms the depression of indentation of the vein where the artery crosses the vein, and the apparent "hump" of the veins over the artery where the vein crosses the artery; at this time nodes appear on the larger vessels of the retina.

There may be arterial pulsation and aneurismal dilations may occur. Veins passing through similar degenerative changes are more unsymmetrically filled and tortuous, later becoming engorged, and even obstructed, by the pressure of a crossing artery.

Together with these ophthalmoscopic signs the subjective symptoms will vary from a temporary scintillation scotoma, temporary blindness as from arterial spasm, to headache, asthenopia and progressively, so-called "failing vision," or "weak vision." The corroborative evidence of high blood pressure, together with the concomitant organic lesions in the liver, kidneys and heart, is of great value, but, as has been pointed out by many observers, among them Mr. Gunn, the lesions may occur in the vascular structures of the eye long before they appear elsewhere in the body, so that lack of the above evidence must not mislead.

It may be well here to recall the probable changes in the cerebral vessels similar to those in the retina, and therefore of prognostic value, that the knowledge of the condition of the retinal vessels gives.

The future of the eye that is diseased dependent on angiosclerosis is bright or dark according to the period at which the lesions are discovered. Nothing is more encouraging to the practitioner in ophthalmology than the results obtained from treating conditions discovered at an early date, when the loss of visual acuity is only temporary, amounting sometimes to 90 per cent. and often to more than 50 per cent.; on the other hand, there is no condition more intractable than a disease of the eye due to long standing changes in the vascular structures. To make a well considered prognosis is always a good rule to follow, and especially is it so in these conditions, for experience shows that without the helpful co-operation of the patient in faithfully carrying out the instructions of the physician there can only be recurrences and ultimate failures from any kind of treatment.

The treatment consists in correcting any disturbance of digestion by diet, lavage, cathartics; regulating the blood pressure; by restricting the fluid intake and reducing the quantity of salt ingested (to prevent retention of excess of fluid); by the administration of nitrites and iodids (sodium preferred, because it does not irritate the gastric mucous membrane and because of its affinity for the connective tissue structure of the blood vessels); by the use of pilocarpin; by baths and packs; and the use of strychnia or tincture of nux vomica. Locally the use of hot moist applications of salt or boric acid and of subconjunctival injections and in some cases dionin.

The sequel of angiosclerosis of the vessels of the eye may be any disease that can possibly result from degeneration of the vessels; from a simple retinitis or chorioretinitis, or retinitis circinata to retinitis pigmentosa or hemorrhagica; to cataract; glaucoma with or without hemorrhage; to embolism of the central or branch arteries or to thrombosis of a branch or central vein.

In conclusion I wish to thank the committee for its kindness in giving me an opportunity to present some views regarding conditions of the structures that carry nutrition to the eye, and which in my experience have caused much ocular disease, that is easily and promptly curable in the early stages, but in later stages requires all the thought, patient care and attention that an earnest worker is able to give, to obtain any satisfactory results from treatment.

DISCUSSION.

Dr. Lewis, Buffalo:—This paper suggests a point in prognosis. I find frequently subconjunctival hemorrhages in conditions that precede or accompany atheromatous vessels, and which led me to question what relation they might have to subsequent cerebral hemorrhages. Often these are not indications of deeper changes; a long period of years may follow before any serious hemorrhages occur elsewhere; but when very small hemorrhages occur in the retina in elderly people, they may be followed very shortly by cerebral hemorrhages. The latter are often of great importance in prognosis.

Dr. Alt, St. Louis, Mo.:—The subject was pretty well discussed yesterday, but Dr. Lamb mentioned among the methods of treatment the use of dionin. We all have experimented with it, and I would like to know in what way he thinks it would do any good in this particular class of cases.

Dr. Jackson, Denver, Colo.:—One point in regard to angiosclerosis we might emphasize more. From the writing in general medical literature we have been predisposed to regard it as a general process. Of course to some extent it is general. If we find a certain vessel decidedly affected there is a possibility that others are affected. But it is commonly much more advanced in some vessels than in others. It may often escape observation, because a single vessel or group of vessels may show unmistakable signs of it, when the greater number of vessels show no evidence of the process. I have seen cases in which some one vessel showed decided changes, while other vessels seemed almost normal. On this account there has been uncertainty about the frequency of the process, and about the general prognosis from what is observed in the eye. It is possible to have it quite advanced in the eye, without serious changes in other vessel areas. One sign that seems to be among the earliest is the change in color of the optic disc. It is not definite enough to rely upon altogether, and it is not present in all cases of arteriosclerosis. But in several cases that I have been able to watch for years there was a brick-red, dull, dark color of the optic disc as a first symptom, afterward followed by marked vascular disease.

Dr. Connor, Detroit, Mich.:—Nearly thirty years ago my attention was directed to a series of papers presented to the profession by Dr. Richey of Washington, and while there was less known of angiosclerosis and other questions Dr. Lamb has presented to us, Dr. Richey emphasized especially the fact that this disease was due to retention in the blood, in the body, and in the tissues, of materials that ought not to be there. He emphasized the importance of getting rid of these things by eliminants and preventing their reformation by proper methods of physiological life. That is exactly what the Doctor has put before us to-day as I understand it. I have always remembered that statement; he insisted I had better stop some of my operative work and study these problems. I must say that as the years have passed that idea has been most helpful to me in securing successful results in ophthalmological work, even in refractive and operative procedures. Yesterday I was disappointed because Dr. Greene was unable to present us with the fact as to the causes of blood pressure—but this comes out in Dr. Lamb's paper to-day. There is something underlying glaucoma. Why does it attack sometimes the posterior and sometimes the anterior eye? We may not know that because of the peculiarity of different individuals, but that there is an underlying disturbance of nutrition, I think, is positively certain. And while we have to treat these cases surgically, we fail to grasp the situation if we do not seek the underlying thing that made them. I am sure we are doing great service when we contribute new facts to show what causes these changes and are able with such knowledge to do some things that will prevent the extension of angiosclerosis and all that follows therefrom.

Dr. Greene, Dayton, Ohio:—Dr. Connor has spoken of his disappointment that I said nothing yesterday in my paper as to the cause of arteriosclerosis. If the gentleman will turn to the title of the paper he will see that this was not its purpose. I did not try to discuss the causes of arteriosclerosis or angiosclerosis. My paper was entirely statistical and had no other purpose than to present a sufficiently large number of statistics so that those who are so disposed can draw their own conclusions from them. I think, however, as we are discussing Dr. Lamb's paper, that we can group these cases under four causes: Strain, heredity, malnutrition and toxic action. This is not an original classification of my own, but is gathered from a paper by Dr. Nichols of Washington, which appeared in *The Washington Medical Annals*, July, 1906. Strain, as was shown yesterday, probably has little influence on the class of men I have examined; hard and laborous occupation was shown to be a more important factor. Heredity is another important predisposing factor. I think it a very important one, and reasoning from a theoretical standpoint, it explains why we see such a difference in people; some are old at fifty or even forty, and usually show abnormal blood pressure. I have seen men in the eighties in good physical vigor, but they have had good arteries and low blood pressure. I think, as Dr. Jackson does, that toxic action is the most important factor in the causation of arteriosclerosis or angiosclerosis. He spoke yesterday in the discussion of the relation of this toxic action to cataract, and if I remember correctly, stated that from his observation toxemia was a more imporant factor in the causation of cataract than arteriosclerosis, but if toxic influences cause angiosclerosis, as is generally believed, and are also causes of cataract, I do not see how it is possible to distinguish between their effects in the two conditions. It seems to me that they are so closely related (perhaps as cause and effect) that they cannot be separated and that we are forced to conclude that in the present state of our knowledge of the subject a general toxemia is responsible for the conditions. Further study will be necessary before we are in a position to say whether the primary force of the poisons is expended in inflammation in the coats of the blood vessels themselves and the lens changes are secondary to the nutritional changes brought about by the diseased condition of the blood vessels, or whether the lens changes are primary results of the toxemia and angiosclerosis only a coincidence. While what has been said above probably contains a large element of truth, I am impressed from the statistics presented yesterday, that angiosclerosis is a common cause of senile cataract—perhaps the most common cause. In 500 men I only observed three cases of subconjunctival hemorrhage, and from this experience I would not attach so much importance to it as has been done as an indication of arterial disease. I think another confusing factor is the localized character of the disease. It is not universally distributed throughout the body as we are so prone to think. When certain large arteries are involved the blood pressure may be normal or but slightly raised; not so, however, when the disease is more generally distributed through the smaller arteries; this distribution raises the peripheral resistance and high or pathological pressure may result.

Dr. Young, Burlington, Iowa:—I hope Dr. Lamb will say something about the different kinds of treatment he employs in the early and in the late cases. I would like to have him define exactly what he means by the early cases, because, in my experience, we do not see these angioscleroses until they have become an established fact. They do not come to us until well established. To make the treatment early, I am reminded of Oliver Wendell Holmes' idea, that the treatment should, perhaps, begin with the grandfathers, as heredity seems to play a part.

Dr. Suker, Chicago, Ill.:—It is gratifying to hear Dr. Lamb use the term, angiosclerosis, for the process affects arteries as well as veins, and to a certain extent the lymph vessels as well. The term arteriosclerosis is too limited in its significance. The ordinary conception of the term arteriosclerosis includes that of phlebosclerosis; arteriosclerosis is specific, while angiosclerosis is generic. Pathologists of to-day have conclusively proved that it is an infectious process which causes this connective tissue proliferation that results in the narrowing of the vessel caliber. And, as any infectious disease, either acute or chronic, lays the foundation for an angiosclerosis, the history of every case which presents those conditions mentioned by Dr. Lamb should be accurately taken. As for increase or decrease in arterial tension, it will be variously manifested some time during the twenty-four hours in any type of disease which is of an infectious origin. The tension in a way is proportional to the amount and virulency of the circulating toxin. When suspicioning angiosclerotic changes in the fundus it is advisable to examine the same under varying intensities of light; begin with a low illumination and gradually increase the same until the maximum amount is reached. Many of the incipient angiosclerotic fundus changes are not at all visible with a maximum amount. In such cases with moderate illumination a preretinal haze is frequently detected, which could not be demonstrated when using an intense illumination. With varying intensities of light the disc can be much more accurately studied and the minute changes spoken of by Dr. Jackson readily noted, which would otherwise have escaped. The irregularities in the chorioid and retina, frequently due to angiosclerotic processes, are not as readily discernable by intense as by moderate illumination. Bearing these facts in mind, not a few of the amblyopias in young children formerly thought to be congenital, will be shown to be due to stationary angiosclerotic processes in the fundus vessels having resulted from a previous acute infectious disease. The angiosclerotic process does not seem to show any preference for any particular set of fundus vessels. However, the peripheral vessels are seen to be most often involved and manifest the earliest changes. Frequently where an artery crosses a vein and compresses it, in that crotch small nodules of transudates are to be observed. Many of the apparent insignificent fundus changes, associated with moderate hazy vision, are the direct consequence of the active or passive hyperemia caused by the angiosclerosis. The changes here are of the nature of infiltrations or transudations with more or less deposition of fine colloidal material. It has been my observation that conjunctival hemorrhages are as great, if not greater, prognostic import than the minute retinal hemorrhages. The retinal vessels being end arteries under high arterial tension would be among the first to yield when affected with angiosclerosis—while the anastomosis of the conjunctival vessels is very profuse, the circulation tension is more uniformly divided, hence, if hemorrhages do occur they are very significant of a disseminated sclerosis involving the larger vessels. As to the blood pressure, it is very important to observe the retinal pulsation while the head is erect and again when fully flexed upon the chest. If there is any angiosclerosis, then the pulse beat is accentuated with the head flexed and the blood pressure in the temporal arteries higher than when the head is erect. This difference in the pressure and pulse is highly significant of a cerebral vascular sclerosis. At times with a rather diffuse fundus angiosclerosis the picture of an albuminuric retinitis is observed, and must be carefully differentiated from the true nephritic albuminuric type. In such cases a detailed urinalysis is obligatory. The former type is quite prone to improvement, while the latter is usually progressive in character.

Dr. Lamb (closing discussion):—I see I selected a popular subject. In regard to Dr. Lewis' differentiation, I think the retinal hemorrhage is much more indicative of an arteriosclerosis in the vessels of the brain than is the conjunctival hemorrhage. The vessels are closely associated and it is much more natural that these two should be associated. The conjunctival hemorrhage, on the other hand, is more significant of a tendency to kidney lesion, which is far away, and we can only say by experience that we have seen a great many of these cases develop Bright's disease later. Dr. Alt asks about dionin. I only use it once. When I find a condition that I think threatens further trouble, as you might say, in advanced stages, I use it to produce a temporary reduction of tension and then give cathartics. I give it to reduce the intraocular tension, simply as a temporary matter. Dr. Jackson's remarks regarding his findings with the ophthalmoscope, I want to corroborate. I had overlooked it for several years, and probably the rest of you may have, but follow a case for several years, have it come back to your office, whenever you see this brick-red disc, and in four or five or ten years you will see the difference. That is the only way you can infer this to be a beginning condition, because you see other cases that have gone through the same stage. Dr. Suker said all I would care to say, had the time of my paper permitted. In the early stages I use sodium iodid or perhaps a little tincture of nux and see that the bowels are open, and I usually find it advisable to go over the entire system, not only, as Dr. Suker says, trying to find out the infectious diseases, but the condition of the kidney, the heart and the blood vessels. I give the iodid in 5 and 10 drop doses and look for a result inside of a week as a rule. In three weeks we usually get good results, even where vision has dropped 50 per cent. In six weeks I have had good result where it had been reduced 90 per cent. I would probably use a little more drastic measures in a case like that than in milder cases. I thoroughly believe in the toxin theory of infection in these cases and its effect on the blood-vessel wall. I want to say one or two things in regard to blood pressure. I think it good corroborative evidence. It is not to be relied upon altogether. Your normal blood pressure and mine are entirely different. The patient with consumption has an entirely different blood pressure, we know. With forced feeding, and immense quantities of milk, it will run to 130 or 150, when there is no doubt that the blood pressure for these cases normally is below 100. The sooner we realize that the blacksmith on the one hand, and somebody else on the other has a different pressure the better off we will be. Each has a blood pressure which is normal for himself or herself, occupation, environment, and condition of health being duly considered. Iodid of sodium seems to have a selective action for connective tissue formation in a great many cases; it has as specific an action as has mercury in syphilis.

GLIOMA RETINÆ, WITH REPORT OF A BINOCULAR CASE CURED.

CHARLES LUKENS, M.D.

TOLEDO, OHIO.

On June 28, 1903, I was consulted about Wilmot G., aged 9 months. His parents had noticed that his eyes were not quite alike for the past three or four months and that they "could see into his head," as they expressed it, in his left eye, and they thought the eye was blind. Thirteen years previous to this date a brother of the father of this child passed through a glioma experience with his own child, the growth recurring a few weeks after enucleation of the eyeball, with the usual termination. This brother and his wife noted a similar appearance to that of their own unfortunate child, and thus the parents were early alarmed and sought professional advice at a time when nine-tenths of the laity would have appreciated nothing abnormal.

Upon examination the child was found to be well nourished and seemed perfectly healthy, there being no pain or discomfort symptoms. His left eye at times diverged, and upon the child's attempting to fix, the left eye manifested a sort of nystagmus. The cornea of the left eye was clear, the pupil was normal in size, the crystalline lins was clear, and back of the lens was a light yellow reflex, across which blood vessels were seen. The tension was normal.

The right eye was not examined carefully at this time, but was apparently healthy. Two days later the left eye was enucleated. The optic nerve was not swollen, although I failed in getting out as much of it as was desirable. The socket healed kindly in ten days and has remained quiet ever since. The eyeball was hardened in a weak formalin solution and cut. The vitreous chamber was half filled with a whitish mass, growing from the posterior half of the eye and extending forward to the posterior surface of the lens. Upon microscopic examination this was found to be a true glioma, springing from the nerve fiber layer of the retina. The optic nerve was not involved and the growth was entirely intraocular.

The child, although well nourished before the enucleation. now became quite robust, and nothing further of note occurred until Dec. 6, 1905 (two years and five months after the operation), when the father wrote me that for three months they had noticed "a light spot in the baby's eye, but that it did not seem to affect his eyesight until one week ago, since which time he can scarcely see to get around."

Examination one week later revealed the following conditions: The eyeball was normal in appearance, the cornea and aqueous humor were clear, the pupil was normal in size and responded to light. A light yellow reflex was seen both in and down, back of the pupil. The center of the retina was functionating, and he could recognize objects when directly in line, the growth encroaching on the visual line from the sides. The tension was normal.

First eye.

Operation was advised, but the parents would not consent to remove the eye while any sight remained. On Jan. 21, 1906, the child had been blind, except light perception, for four days. Enucleation was now performed, removing 9 mm. of the optic nerve, which was not swollen. The socket healed well, considering the amount of mutilation necessary in order to get as long a piece of optic nerve as possible. The child, while apparently not sick before the operation, now very noticeably increased in vigor. He was last examined on July 14, 1907. Both orbits were normal in appearance and to palpation, the child was thin in flesh, but there were no symptoms of brain envolvement. My last history of him was Sept. 17, 1907, when his father reported him well.

On cutting the second eyeball the growth was found to completely encircle the vitreous chamber, half filling it. It was cream color and lobulated. It seemed to start from two foci, the larger growth being on the nasal side, the lesser on the temporal side. These lobules pushed out into the vitreous space until they nearly met, obscuring the macula, which was the only area of the posterior, five-sixths of the retina not apparently involved by the growth.

The microscope revealed glioma. The macular area showed the latest involvement, where both granular layers showed beginning glioma proliferation. The optic nerve showed infiltration in its scleral portion only. The nerve for 9 mm. beyond the ball was free from tumor cells.

Glioma is the only primary neoplasm of the retina. It is confessedly a rare disease and is limited to childhood. Statistics of different observers show its occurrence, ranging from one case in every two thousand[1] ophthalmic patients to one in twenty thousand.[2] This affection has been known and described more or less imperfectly for several centuries, although under varying names, its present name dating from Virchow‘s glia theory. Microscopically the neoplasm closely resembles round cell sarcoma, and many general surgeons and pathologists, including Green,[3] Wyeth,[4] Keen and White,[5] Gowers,[6] Senn[7] and others, classify it as a variety of round-cell sarcoma. Warren[8] classifies it under benign tumors and says that so-called metastases into distant organs are probably sarcomata, although he does not state how we can have a metastasis of sarcoma from a benign tumor. Ziegler[9] says that "glioma of the retina is closely related to glioma of the brain, from which it differs in this particular, namely, that it gives rise to metastases in various parts of the body and tends to invade contiguous tissues." Wintersteiner[10] believes that the rosette formations found in about one-third of all cases are the key to the growth. These contain cells resembling rods and cones. He assumes they are the start of the growth, from the outer (epithelial) layer of the retina, and adopts Flexner's[11] term, neuroepithelioma. Alt[12] considers these rosettes to be tissue enclosures. Mallory[13] and Verhoeff[14] both declare that malignant growths of the retina are not glioma, because they can not demonstrate glia fibers in these tumors with a reliable neuroglia stain (Mallory's). Verhoeff[14] further states that "the tumor ordinarily known as glioma retinæ is really composed of nervous elements in an embryonic state,"[15] and suggests the term "neuroma retinæ malignum."

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[1] Schöbl: Diseases of the Retina, Norris and Oliver's System of Diseases of the Eye, vol. ii, p. 571.

[2] Fejer: Pester Medicinische-Chirurgische Presse, July 29, 1902.

[3] Pathology and Morbid Anatomy, 1889.

[4] Text-Book on Surgery, 1890.

[5] American Text-Book of Surgery, 1892.

[6] A Manual of Diseases of the Nervous System, Second American Edition, 1893.

[7] The Pathology and Surgical Treatment of Tumors, 1895.

[8] Surgical Pathology and Therapeutics, 1894.

[9] Text-Book of Special Pathological Anatomy (English translation, 1897).

[10] Wiener Klinische Woch., 1894, xxvii, p. 493.

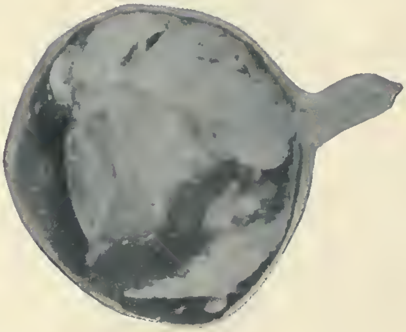
[11] Johns Hopkins Hosp. Bulletin, 1891, p. 115.

[12] American Jour. Ophthalmology, St. Louis, 1903, xxi, p. 257-277.

[13] Reference Hand-Book of the Medical Sciences, 1903.

[14] Rare Tumor from Pars Ciliaris Retinæ, etc., Transactions American Ophthalmological Society, 1904.

But the literature is voluminous and discordant as to the nature of this growth. We may profit more at present by considering certain therapeutic dicta, which are being surely established. All unmolested cases of glioma retinæ inevitably die, and many cases die in spite of operation. The neoplasm spreads both by continuity of tissue and by metastasis. As with other malignant tumors, the tissue invaded may change the character of the growth. Many earlier observers advised against any operation, believing the disease to be incurable. Hirschberg and von Graefe corrected this idea, the former reporting five cures out of twenty-seven cases. Later observers give a better percentage of recoveries. Wintersteiner gives 13 per cent. of recoveries, if two years be taken as the period to establish a cure. Jessop[16] very justly corrects Wintersteiner's statistics, which included non-operated cases and death from other cases; his corrected statistics of Wintersteiner give 20 per cent. of recoveries in two years.



Second eye.

Jessop[16] contends for the classification of glioma into two stages only, viz., intraocular and extraocular. He analyzes forty-seven fatal cases and thirty-four cured ones and found that in only one of the fatal cases was the growth apparently purely intraocular, and of the thirty-four recoveries all were intraocular except six, in which the nerve was affected. He therefore, strongly advises that an eye should never be excised for glioma without taking as much of the optic nerve as possible, from 12 to 15 mm. (Fifteen mm. of optic nerve of a baby is not so easy to get.)

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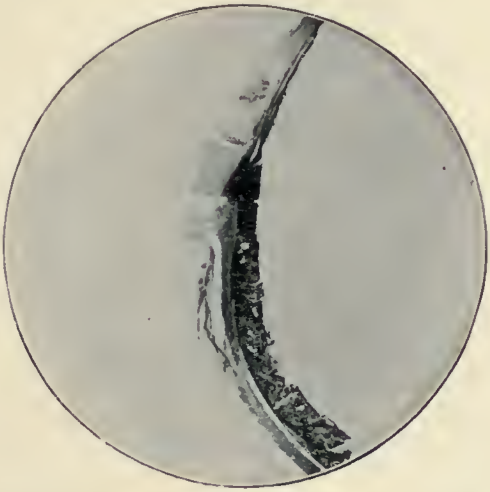
[15] This was pointed out by Treacher Collins, Researches, London, 1896.

[16] St. Bartholomew's Hospital Reports, vol. xxxviii, p. 159.

A recent report of Professor Hirschberg[17] bears out Jessop's observations. He states that he has operated sixteen times for glioma, in ten of which the growth was limited to the retina and in none of these was there a recurrence. Of the other seven, four recurrences were known to have taken place. He concludes that the prognosis of glioma is good if the operation be performed within ten weeks from the time of the appearance of the whitish reflex. C. R. Holmes[18] operated on six eyes in five patients, four of them in the intraocular stage, with four recoveries, although in one of his reported recoveries the second eye became involved three years later and killed his patient. Personally I have seen seven glioma patients, six of them were operated on, and in one operation was refused. Of these, five were in the extraocular stage, the nerve being involved, and in one the growth had passed through the sclera. The other case, the one of double operation detailed above, is the only one living, and both of these eyes were excised during the intraocular stage.



Section showing tumor infiltration of optic nerve in scleral opening, second eye.

These later observations show no less malignancy, but strongly indicate the almost certain curability of the disease so far as the affected eye is concerned, if operated on before the growth had passed the confines of the eyeball. It is impossible to determine this before the enucleation, although the advent of inflammatory symptoms is strongly presumptuous that extraocular extension has taken place. Even then an operation should not be denied, as it offers the only chance for cure and will relieve a hopeless case from weeks of suffering.

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[17] Centralblatt für praktische Augenheilkunde, April, 1904.
[18] Journal A. M. A., March 28, 1903.

The diagnosis of glioma should not be difficult if seen before the media become clouded. Consider the age, the light yellow reflex, the damaged eyesight, and exclude pseudo glioma, which usually results from an injury, or pus elsewhere. Also rule out simple detachment of the retina, and sarcoma of the chorioid. The latter is never seen before the second year and rarely before the tenth (Ziegler). Tubercle of the chorioid, sub-retinal cysticercus and congenital malformations have been mistaken for true glioma. Where inflammatory symptoms have made their appearance the diagnosis becomes more difficult. But even then the parents will be able to give valuable information as to the earlier history. They usually remember having seen the bright yellow reflex, particularly if questioned on this point. In doubtful cases with a blind eye, operation should be performed, for the chances greatly favor its being glioma.

CONCLUSIONS.

First.—Be on the lookout for glioma of the retina; failure to diagnosticate usually depends upon failure to consider this disease.

Second.—Operate as early as possible after the diagnosis is made, if possible within ten weeks from the first appearance of the whitish reflex, and cut the optic nerve as near the apex of the orbit as possible.

Third.—If the second eye becomes involved after one eye has been successfully removed (i. e., no recurrence) this eye should likewise be removed.

Fourth.—When in doubt as to diagnosis, operation is usually conservative surgery.

Fifth.—Recent observations indicate that glioma retinæ is curable in 90 to 95 per cent. of cases, if properly dealt with.

DISCUSSION.

DR. ALT, St. Louis, Mo.:—I had two cases of glioma in which the enucleated eyes showed the optic nerve already invaded by the growth for quite a little distance behind the sclerotic, but no recurrence has taken place. One of these children was operated on about seven years ago and the other four years ago. The latter child had an invasion of the optic nerve 4 mm. beyond the lamina cribrosa. I told the parents that I was afraid it was too late and a recurrence or metastases might be expected, but the child is still healthy and well. She was about 4 years old when I enucleated the eye. I have since seen her at different times when I prescribed glasses for the healthy eye. I had just such an experience. A child was brought to me with double glioma, but the parents refused the enucleation of both eyes, preferring the child to die. Of course this was their privilege, and they stuck to it, although I pictured to them what the poor child would have to go through before death would come to its relief.

Dr. Giles C. Savage, Nashville, Tenn.:—Two years ago about this time a little patient was presented to Dr. Price of Nashville in which he diagnosticated a glioma in one eye. The parents were prevailed upon to have the eye enucleated. Between then and the following February it developed in the other eye, and it was removed. The little patient was seen about three days ago; it is 3 years old now, lively as can be, and no evidence of return. Whether there will be a return or not we do not know. I do not think it should be a hard matter on the part of any man, aided by a consultant, to induce parents of children with glioma to have the second operation done if it appears in the second eye. I can not see how they could reject it when they know death is the only other alternative.

Dr. Fridenberg, New York:—Without appearing hypercritical, I would suggest that the title of the paper is misleading, because "Glioma Retinæ with Binocular Case Cured," sounds as though the eyes were saved, whereas they were lost. Dr. Lukens calls attention to the fact that operation results in the saving of the life of the patient in a large number of cases. He is to be congratulated on the fact that the patient was saved where the disease had invaded both eyes, but the glioma retinæ was not cured.

Dr. Minney, Topeka, Kas.:—I have had two cases, one of nine years and the other now of thirteen years standing, where the children are well. I met with difficulty after I had diagnosticated the same trouble in the other eye. They would not let me operate, but they had learned enough when the sight was lost to have somebody remove the other eye. This was thirteen years ago and I have followed the case since. The other case is of nine years standing and it did not appear in the other eye. I would like to know why these cases remain cured, whereas in other cases of these small cell sarcoma in aged persons they will return in from two to five years. I have had two or three of these cases and they do return in other parts of the body and then die. Why is it that such is the case?

Dr. Lukens, Toledo, Ohio:—In defense of my use of the word "cured" in my title: The patient was cured of the disease the same as the general surgeon cures cancer, by excising the affected organ. This is entirely in accordance with the established use of the term. As to obtaining consent for removing the second eye; in my particular case the first cousin of this child had gone through a glioma experience and these parents knew as well as I did that it was inevitable death without operation; hence I had no trouble. One colleague asks why gliomas are cured and sarcomas in other regions recur in from two to five years after removal. Glioma is not sarcoma, and the history of these cases is that they are not found after the age of eight or ten years, and that if one recurs after operation, it is almost surely within a few months, usually within a few weeks, although a glioma may appear *de novo* in the other eye, as in my case, several years after operation. But, where no retina exists for a new growth to start in, and twenty months have elapsed with no recurrence, I feel justified in claiming a cure.

ON THE MUSCULUS DILATATOR PUPILLÆ.

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ST. LOUIS, MO.

A number of the more recent text-books on ophthalmology speak of the dilatator muscle of the pupil as an undoubted and definitely proven entity. Some even give illustrations which plainly show that the author has never seen the actual conditions. There are usually two well and equally developed layers of epithelial cells on the posterior surface of the iris which are separated from the iris tissue proper by one layer or several layers of spindle cells which run from the iris periphery to the sphincter pupillæ. The illustrations look so nice and simple, and from the appearance of the tissues it is so easy to recognize what is intended to be shown, that any one not particularly well acquainted with the subject must, indeed, wonder why so much time should have been wasted in studying and writing and fighting about the existence or non-existence of this dilatator muscle, when everything about it is apparently so plain.

Yet, that such drawings and descriptions are probably not representing the true relations, and that there must be peculiar conditions which do even to this day not allow of an absolutely final decision, although we seem to be very near to it, may become clear even to the uninitiated, when remembering that K. Muench[1] could state that up to 1903 no less than 216 articles had been published which dealt with the anatomical and physiological side of the dilatator pupillæ question, and, up to 1904, no less than 617 in which the clinical and pathological aspects of this question were discussed.

From this statement it is clear that a question which had been studied more or less unsuccessfully by so many ardent workers in the field of science, must offer especial difficulties. These difficulties were particularly great before methods had become known by which the pigment contained in the protoplasma of the cells could be bleached and eliminated, and thus an unobstructed view of the cell structures could be obtained.

Quite naturally, the discovery of such methods by Collins, Griffith, Alfieri and others gave a new impetus to the study of the dilatator muscle, and in consequence a further number of papers on the subject have appeared more recently. It may be interesting to consider some of these more especially.

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[1] Graefe's Archiv., vol. lxiv, p. 339.

K. Kiribuchi published a paper[2] on "Studies Concerning the Elastic Tissue in the Human Eye, with Remarks on the Musculus Dilatator Pupillæ." He bleached the iris and then stained for elastic tissue according to Weigert's method. (In parenthesis, I may say here, that he found the iris tissue free from elastic fibers, except along the blood vessels and in the interstitial tissue between the fibers of the sphincter pupillæ, statements with which the results of my own examinations agree perfectly.)

Concerning the dilatator he says the following: "The so-called limiting membrane (*Bruch's Grenzmembran*) which lies in front of the pigment epithelium layer is stained slightly blue, by Weigert's method, like the musculus sphincter pupillæ, while other so-called elastic membranes, like Descemet's or the hyaloid membrane of the chorioid (lamina vitrea) are stained deeply, just like elastic fibers. Since this stain is an absolutely elective one, it follows that the limiting membrane of the iris must be of a nature which differs from the elastic membranes. According to Henle, Merkel, Luschka, Jeropheeff and others, this limiting membrane consists of unstriped muscular fibers, while their antagonists, Gruenhagen, Hampeln, Schwalbe, Michel, Koganëi, Fuchs and others describe it as a structureless vitreous membrane with elastic properties or as a tissue midway between elastic and connective tissue. According to my (Kiribuchi's) examinations, the limiting membrane is not an elastic membrane; yet, as is well known, the posterior part of the iris is more elastic than the anterior one, and, since, when the pupil dilates, the limiting membrane is not thrown into folds, it must possess a great degree of elasticity. Since, furthermore, a wide dilatation of the pupil can not be explained by the simple contraction of the blood vessels, and since there is no other organ besides this limiting membrane to which a pupil dilating force could be ascribed, this membrane, if it is not elastic, must be muscular. Like K. Grunert, I could see in my bleached specimens which were stained after Van Gieson, or with 3 per cent. hematoxylin, a layer of spindle cells with long rod-like nuclei. It seems, therefore, that a dilatator muscle is present in the iris."

A number of the more recent writers do not hesitate to declare, like Kiribuchi, that the limiting membrane (*Bruch's Grenzmembran*) is undoubtedly the dilatator pupillæ. However, the descriptions which they give of the conditions as they appear to them differ so materially in essential points that it looks as if, paradoxical as it seems, a better opportunity to see had actually increased the difficulties and made things worse confounded.

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[2] Knapp's Archiv., vol. xxxviii, p. 117, 1898.

A. Szili, Jr., has been fortunate enough to have sufficient fetal material at his disposal to study the development of the muscles of the iris. The results of his extended studies are published in a paper (Beitrag zur Kenntniss der Anatomie und Entwicklungsgeschichte der hinteren Irisschichten, mit besonderer Berücksichtigung des Musculus sphincter pupillæ des Menschen).[3] He comes to the conclusion that "the musculus sphincter pupillæ in man is an epithelial muscle. It develops about at the beginning of the fourth month from the epithelial cells at the turning point of the two leaves of the secondary ocular vesicle." He further states: "The musculus dilatator pupillæ is an epithelial muscle. It takes its origin from a transformation of the anterior layer of epithelial cells of the iris in the seventh month of embryonic life."

No one has so far been able, it seems, to verify or contradict these statements. Yet the fact that both iris muscles are of epithelial character has, since the appearance of this paper, been tacitly accepted as proven. One point which Szili particularly dwells upon is the question whether one or two layers of epithelial cells cover the posterior surface of the iris. His description and explanation we will return to later on.

Even this apparently simple question is evidently not so easily settled. Grunenhagen, Widmark and others describe a dilatator muscle and two layers of epithelial cells behind it. Grunert thinks that in the adult there is only one layer and that only here and there a few cells of the anterior layer have remained in existence between the dilatator and the single posterior layer. Heerfordt states that the limiting membrane consists of fibers without nuclei originating from a coalescence of the epithelial cells during fetal life. Hotta studied this question[4] on the eyes of anthropoid apes. He found that the limiting membrane in these apes contains no nuclei, but that the muscular nuclei are, so to speak, adherent to the posterior surface of its fibers, and appear rod-shaped and elongated with a contracted and oval or round, with a dilated pupil. He never found two layers of epithelial cells, but doubts the correctness of Heerfordt's opinion, according to which the fetal anterior layer of epithelial cells is transformed into these muscular fibers without nuclei. His own opinion is that the rod-shaped or more rounded nuclei of the so-called anterior epithelial layer must be looked upon as the nuclei of unstriped muscular fibers.

He concludes with these statements: (1) The limiting membrane combined with the nuclei at its posterior surface must be considered as one layer of unstriped muscular plates. (2) What is called the limiting membrane is a layer of the contractile substance of muscle cells or contractile cell plates. (3) The nucleus lies on the posterior surface of and externally to the contractile substance, surrounded by protoplasma. (4) When tho muscle is stretched the nuclei approach the contractile substance and *vice versa*. (5) When the muscular layer is very strongly stretched the nuclei assume the rod-shape and are pressed closely to the contractile substance so that it may appear as if they were lying within it. When the muscle is contracted the nuclei appear oval or almost round.

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[3] Graefe's Archiv., vol. liii, p. 459.

[4] Graefe's Archiv., lxii, p. 250.

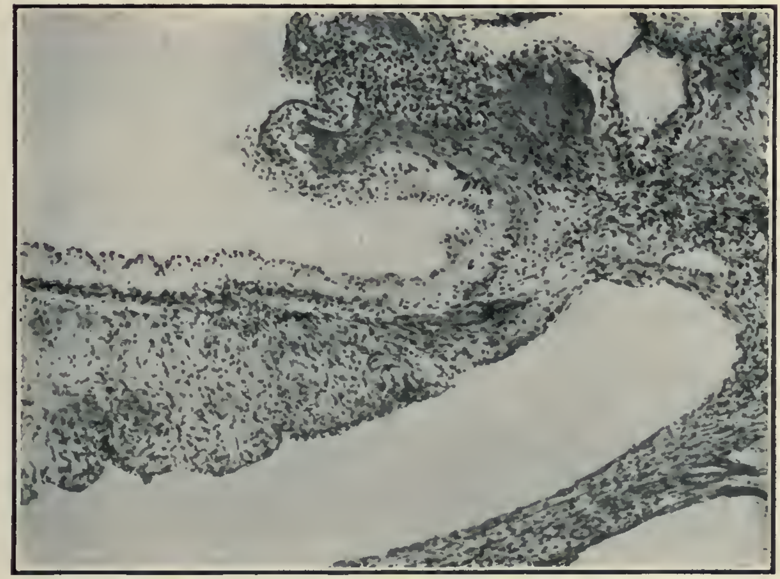

Fig. 1.—Bleached iris periphery and ciliary body; two layers of epithelial cells, and in front, separated in specimen, rod-shaped nuclei of dilatator.

G. Levinsohn, in a very elaborate paper,[5] comes to the conclusion that the posterior surface of the iris is made up of the dilatator layer of closely united unstriped muscular fibers, and two distinct layers of pigment epithelium cells. Juler is of the same opinion, except that he assumes several layers of muscular fibers. Gabriélidès[6] describes a very similar arrangement in the iris of the chicken. Gruenhagen finds a complete posterior and an incomplete anterior epithelial layer behind the dilatator muscle.

A. von Szili, Jr., in his latest paper,[7] severely criticizes Levinsohn's paper, before mentioned, and contends that the results of his own developmental researches had marked a decided progress in our knowledge, while Levinsohn's, in his opinion, are a decided step backwards.

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[5] Graefe's Archiv., lxii, p. 547.

[6] Archives d'Ophtalmologie, March, 1895.

[7] Graefe's Archiv., vol. lxiv, p. 141.

He again refers to the history of the dilatator researches, gives Henle the credit of having been the first to recognize that Bruch's limiting membrane (Grenzmembran) is a muscular organ and divides the different authors and their opinions into five groups:

1. Those who assume an elastic membrane in front of one layer of pigmented epithelial cells—Bruch (Bruch'sche Grenzmembran).

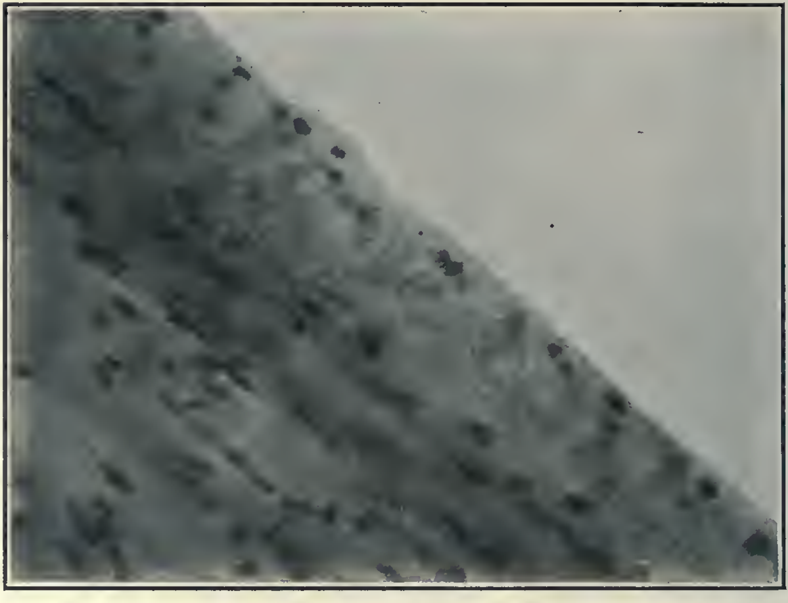

Fig. 2.—Bleached. From the part of the iris midway between periphery and pupillary edge. Two distinct layers of epithelial cells, in front of which the continuous layer of rod-shaped nuclei of the dilatator.

2. Those who assume a single epithelial layer and accord to Bruch's membrane a muscular character—Henle, Merkel and Iwanoff.

3. Those who accept two pigment epithelial layers and in front of them a non-muscular fibrillar or elastic limiting lamella which is sometimes described as a structureless vitreous membrane—Gruenhagen, Hampeln, Boé, Schwalbe, Michel, Eversbusch, Koganëi and Fuchs.

4. Those who describe two pigment epithelial layers and give to the limiting membrane a muscular character—Dostoiewsky, Widmark and Juler.

5. Those who, perfectly aware of the fetal existence of two pigment epithelial layers, declare that in the adult only one such layer exists.

He then states that this array of names, with diametrically opposed opinions, all of them based on thorough work and conscientious research, quite forcefully proves that the histological appearances alone do not suffice for a final settlement of the dilatator question. This final solution can only be seen in the results of his own, previously mentioned, developmental studies. Having tried to prove Levinsohn's statements false and untenable, he now comes to the following conclusions:

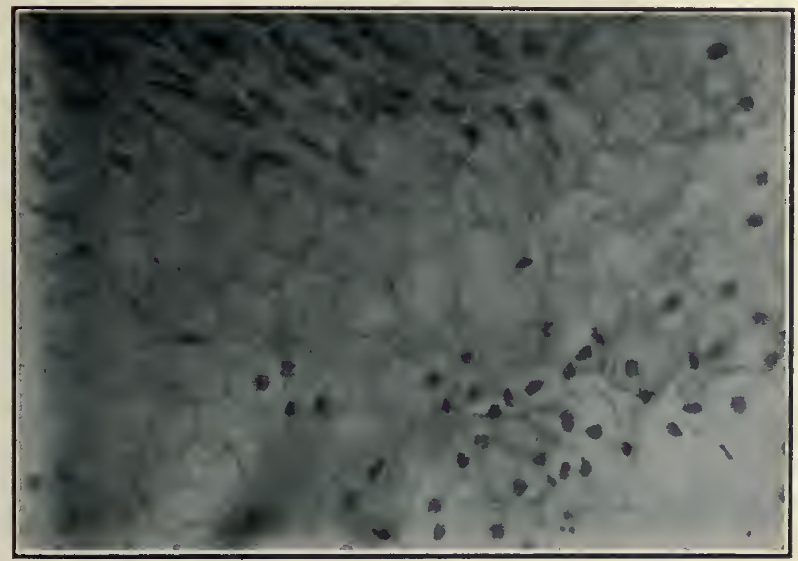

Fig. 3.—Bleached. Nearly flat section. Epithelial cells below, rod shaped nuclei of dilatator above.

"1. The muscle fibrillæ of the dilatator pupillæ in man are formed in the embryo as an intracellular differentiation in the basal parts of the cells of the anterior epithelial layer.

"2. The posterior covering of the adult iris in the area of the dilatator consists from within outward (*a*) of one layer of well-formed epithelial cells and (*b*) of one layer of oval nuclei surrounded by more or less protoplasma and in front of these a fibrillar layer.

"3. Based on the developmental history, we are forced to declare the anterior layer of nuclei and the fibrillar layer in front of it in the adult as closely connected and to call the two together the dilatator pupillæ muscle."

To these references may be added a paper by K. Muench,[8] who assumes that all of the stroma cells of the iris are of a muscular character.

I am now coming to my own studies concerning the dilatator. When I was a student of medicine we were taught that the muscular nature of the sphincter pupillæ was an undoubtedly proven fact, that the dilatator had as yet not been demonstrated, but was "a physiological necessity." But even then some authorities thought they had seen the dilatator muscle, while others as stoutly denied its existence.

In the studies concerning the histology of the human eye which I began during my student days, I was attracted by this problem. However, like others, I was absolutely unable to convince myself of a muscular layer in the iris which might be looked upon as the ardently sought for dilatator pupillæ. The question seemed to be solved by Julius Arnold's opinion that the unusually thick muscular coat of the iris arteries running in a radiary direction from the periphery of the iris towards the pupillary margin, when contracting, would dilate the pupil.

Fig. 4.—Bleached. Flat section of dilatator. Rod-shaped nuclei lying at different levels.

The only tissue which in some way reminded one of a muscular coat was a layer of apparent spindle cells with more or less rod-shaped nuclei which could be seen on the posterior iris surface when the pigment epithelium was gently scraped off. However, these cells contained pigment and with the staining material then in use gave no reaction which would have proven them to be of a muscular nature, nor was their shape exactly as slender as that of unstriped muscular cells elsewhere in the body.

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[8] Graefe's Archiv., lxiv, p. 337.

To my astonishment, C. Faber (Der Bau der Iris des Menschen und der Wirbelthiere) in 1876 described these very cells unhesitatingly as the dilatator muscle.

Although I took up the subject a number of times, I always had to acknowledge that I could not find an undoubted dilatator pupillæ.

After T. Collins had introduced his method of bleaching the iris pigment I worked diligently in order to perfect myself in its application and soon succeeded in getting good specimens. Now, I thought, there could no longer be any difficulty in proving the existence or non-existence of the dilatator muscle. I have since used Alfieri's method with even better results.

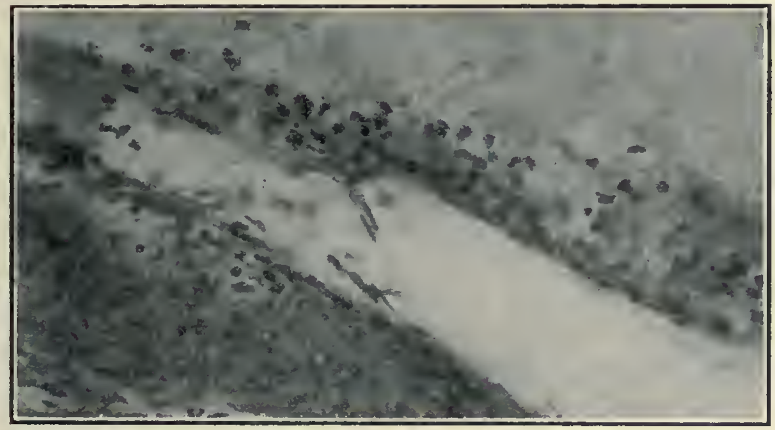

Fig. 5.—Bleached. Posterior epithelial layers below, separated from iris tissue above. Several long, slender, spindle-shaped cells with rod-shaped nuclei have become isolated. Dilatator fibers.

Such bleached specimens show in meridional sections the posterior surface of the iris covered with epithelium which forms two layers at the iris periphery, where it joins the two epithelial layers of the ciliary body, and at the pupillary margin behind the sphincter pupillæ (Fig. 1). In the intermediate part there are marked differences in specimens from one and the same and from different eyes. There is mostly only one layer, but in different places, more often in some, less in other eyes, a double row of epithelial cells is found (see Fig. 2).

If, according to Szili, the dilatator muscle is formed by transformation from the cells of the anterior layer of pigment epithelium cells, it is astonishing that in so many places this transformation has apparently not taken place.

In front of these pigment epithelial cells lies a continuous row of spindle cells with a mostly rod-shaped nucleus, which, when the section is exactly at right angles to the surface appears double or triple, because the nuclei do not always lie at the same level. Yet sections made on the flat which, if successful, may show larger portions of this layer alone, seem to make it certain that there is only one layer of such rod-shaped nuclei. The outline of the single cells to which they belong I have never been able to produce by the different methods of staining. Yet not infrequently a separation of the posterior layers takes place by pressure on the cover-glass, and then long slender spindle cells with a rod-shaped nucleus become here and there isolated and visible (Figs. 3, 4, 5). Where there are two layers of pigment cells, in spite of Szili's statement, I always find their oval or round nuclei as well as the rod-shaped ones of the layer in front of them.

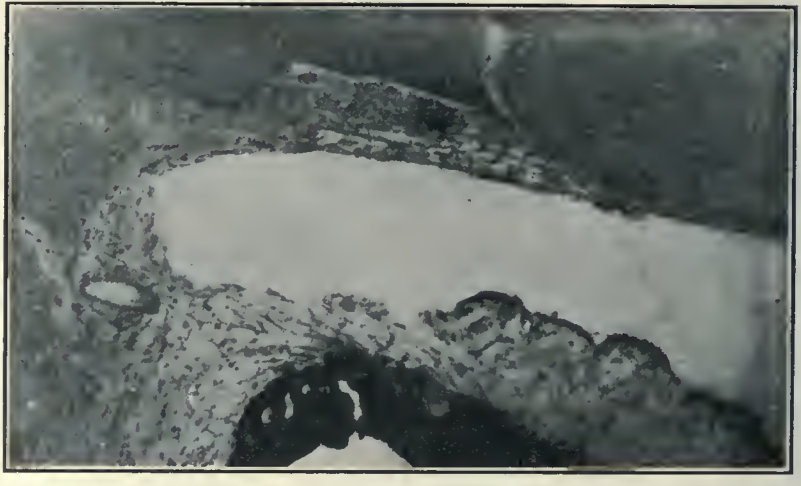

Fig. 6.—Fan-shaped tendon of dilatator. (Accidentally this section shows well the connection of the venous plexus with the meshes of the ligamentum pectinatum.)

In front of this layer of rod-shaped nuclei, when the posterior layers have been split off, a small amount of an amorphous or slightly granular substance is occasionally seen which takes on a little deeper tint; in this, also, I have found invariably some rod-shaped nuclei. It is evidently a part of their layer adhering to the iris tissue proper, and I suppose this corresponds to what Szili describes as the layer of fibrillæ which, according to him, are formed by an intracellular transformation in the cells of the fetal anterior epithelial pigment layer and lies in front of the nuclei.

The layer of rod-shaped nuclei is closely applied to the anterior pigment epithelium layer except at the iris periphery, where it reaches farther outward than the latter and is separated from it by some intervening connective tissue.

Taking all things into consideration, we must probably now affirm that this single layer of slender spindle cells with a rod-shaped nucleus forms the muscular dilatator pupillæ. The peculiar part of it is the close union of the cells with each other, so that their outlines are invisible and the individual cells can not as a rule be recognized.

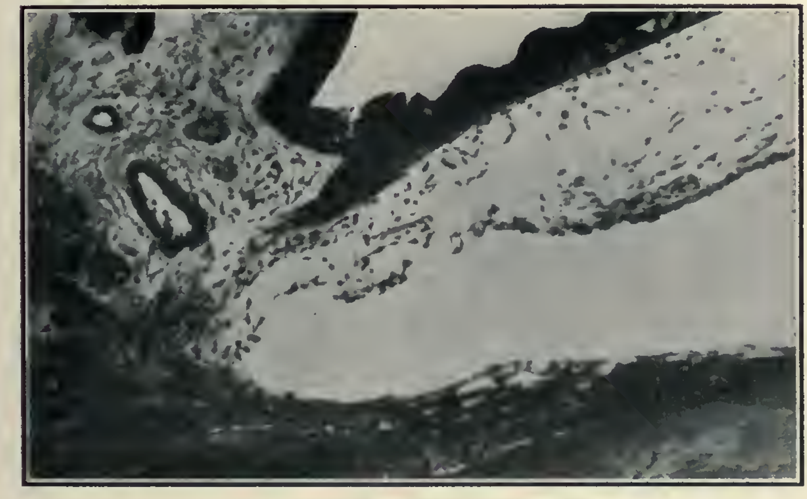

Fig. 7.—Band-like tendon of dilatator.

At the pupillary edge the dilatator joins the posterior surface of the sphincter muscle. At the periphery of the iris, fibers starting from the dilatator spread fan-like out and backwards into the connective tissue of the ciliary body (sometimes I have thought I was able to follow such fibers into the anterior part of the ciliary muscle), or in other sections, such fibers form a more or less sharply defined band which, passing in front of the circulus iridis major, is lost in the inner layers of the ligamentum pectinatum (Figs. 6 and 7). The latter form of tendon, if we may call it so, has been previously described by A. E. Ewing,[9] who thought this to be the only form of peripheral attachment of the dilatator and to be conspaces between the ciliary processes. If his opinion is correct the fan-shaped termination may, perhaps, apply more to the ciliary processes themselves. I have been unable to satisfy myself as to this point.

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[9] Graefe's Archiv., xxxiv, part 3.

Granted, then, that what we have seen and described is the musculus dilatator pupillæ, it appears that it is rather a weak muscle in comparison to the sphincter pupillæ. It seems, therefore, very probable that its action is enhanced by the contraction of the iris arteries and, perhaps, by the relaxation of the sphincter muscle whenever a dilatation of the pupil takes place.

DISCUSSION.

Dr. Fridenberg, New York:—The histological demonstration that Dr. Alt has given us is corroborated by the experimental investigation in regard to the supply of the dilatator by Meltzer-Auer of New York, who finds in case of paralysis of the sympathetic a lack in innervation of the stimulating branch of the dilatator, so that the instillation of cocain, acting as it normally does on the nerve, fails to have any effect on the pupil. Another point is that from the cervical ganglion there runs an inhibiting branch to the dilatator pupillæ, and if that branch is paralyzed, the dilatator is free to respond to stimulus of drugs that ordinarily do not affect it. When you have paralysis of both fibers adrenalin will produce maximal dilatation. After extirpation of the ganglion, we have to bear in mind that if the inhibiting fibers are removed adrenalin becomes a mydriatic, and if we are going to extirpate the ganglion for glaucoma, we should leave posteriorly the inhibiting fibers. These experiments were done on rabbits, and the clinical observations made on the human being.

Dr. Reynolds, Louisville, Ky.:—According to my understanding, all muscular fiber must have an investing sheath. In fact, that is one of the characteristic points of differentiation to be considered in studying the stroma of the iris. It may not, at all times, be easy to differentiate. the yellow, elastic, connective tissue fiber, with its attached pigment cells, from the divided continuations of the periphery of the posterior elastic layer of the cornea, which are deflected upon the surface of the iris to form the pectinate ligament; and, as they continue toward the pupil, continue dividing up into hair-like lines, until they are attached by fusion to the sheath of the constrictor pupillæ muscle. Regarding these filiform bodies as continuations of the pectinate ligament of the iris, it is easy to understand the difficulties encountered in the search for radiary muscular fibers. The stroma of the iris, with its cylindric filiform and nucleated spindle cells, some with anastomosing processes, all mixed in with the pigment cells, the blood vessels, and nerves, and the minute system of lymphatics, presents such a complicated arrangement as to have baffled all previous search for radiary muscular fibers in the iris. I have not been able to satify myself that any definite muscular characteristics have been shown to dwell in any cells or fibers passing in a radiary direction through the iris. In all the descriptions which I have read of the radiary muscular fibers of the iris, there is an absence of positive and definite differentiation between the connective tissue elements of the pigmented portion and the filiform continuations of the pectinate ligament.

Dr. Alt, (closing discussion):—I spoke of fibers which appeared to form the tendon, not the dilatator fibers themselves. Those I tried to show in the photographs, particularly when the posterior layers were removed from the tissue proper. Those fibers that Dr. Reynolds speaks of are connective tissue fibers and do not take up the elastic tissue stain.

REMARKS ON VIBRATORY MASSAGE IN EYE DISEASES.

LEARTUS CONNOR, A.M., M.D.
DETROIT, MICH.

Finger massage of the eye is as old as medicine. It has been used direct, to the conjunctiva, or indirect through the lids, the movements being vertical, horizontal, circular or radial. While employed without or with medicaments, usually some ointment—most frequently a mercurial—is placed in the conjunctival sac or on the lid. It is not easy to separate the benefit of the mechanical movements from that of the medicine used—probably the movements with or without pressure are most important. Every ophthalmologist uses this treatment, in some form, with varying degrees of frequency and satisfaction in chronic corneal or conjunctival disease.

In the treatment of obstructions in the retinal vessels, Dr. Würdemann urges deep pressure, with movements on the eyeball, so hoping to dislodge the obstacle and restore vision.

Midway between these forms of massage are such pressure movements as affect the ciliary circulation and hasten the escape of lymph from the eyeball, so reducing pressure. It is said to render the operation for glaucoma easier by deepening the anterior chamber, while reducing the tension, and to affect favorably many forms of chronic chorioidal and uveal diseases.

Sometimes good service is done by gently striking the eyeball with the ends of the fingers, more or less rapidly, so getting the effect of interrupted blows.

Massage of the lid margins for blepharitis marginalis and associated affections, with varying strengths of mercurial ointments, is a familiar practice, having the double effect of killing microbes and reducing the swollen tissues.

This introduces the question, "What, if any, addition has vibratory massage brought to ophthalmology?"

To the friction of finger massage, as it passes to and fro, up and down, round and round, with more or less pressure on the eyeball, vibratory massage adds a longer or shorter, slow or quick vibration, starting from the point of the instrument as it touches the eyeball, and passing to every other part, along nerve or blood vessels up to the brain and other adjacent structures. It must thus quicken the outgoing currents of lymph and venous blood, so inviting to the vacant spaces good arterial blood and fresh lymph. It must stimulate the sensitive nerves and provoke vasomotor activity with an accelerated arterial current. It must arouse to increased action every visual or nutritive cell and tend to break up irregular activities in any part of the ocular apparatus.

Like other ideas, much relative to vibratory massage has come from without the recognized profession. As is usual from such sources, the wildest claims have been made for its power, and so it has been discredited in advance of actual investigation by persons of good judgment. In the books and journals within my reach I have found little relating to vibratory eye massage, and this very indefinite.

Compared with "finger eye massage," the vibratory calls for a delicate special apparatus, of considerable expense to install or maintain, and apt to need repairs at inopportune times. Its best operation calls for connection with a street current, thus limiting its use to those having such a current or willing to bother with galvanic batteries. The use of vibratory massage is a large time consumer, mainly in getting the patient in relation with the apparatus, so that the busy doctor must believe that he can accomplish by it something beyond old methods, else he will pass it by.

Vibratory massage has psychic as well as material effects which must be carefully distinguished in estimating their relative value, though each may often aid the other.

Some years since I had a manufacturing electrical company combine in one instrument the means of transforming the street current into each of the forms known to be useful in managing diseases of the eye and related organs. Since then as opportunity presented I have tried to ascertain what, if any, value vibratory massage has over older methods. This apparatus, driven by a one-eighth horse-power motor, has a convenient and effective device for giving vibratory eye massage. It admits a wide range of length, frequency and force of vibration. It gives deep or superficial massage, in any direction, as well as the tips of the fingers, adding to the latter a vibratory movement slow or rapid, long or short, light or heavy, as may be desired.

While the exact effects of this addition to eye massage has never been satisfactorily worked out, certain facts seem reasonably certain. Thus (1) if one places his finger on the eyeball while applying massage, the vibrations are distinctly felt, showing that they reach the entire eyeball and hence the nerves, blood vessels, muscles and lymphatics attached thereto. (2) Most patients recognize a greater clearness of vision and are usually able to read more and easier, as proven by using test types, before and after the massage. (3) The fundus is more distinctly seen by the ophthalmoscope after massage. (4) Varying with the pathological conditions, definite changes can be observed in the central retinal vessels, generally an enlargement of both central retinal veins and arteries. (5) Often the fundus reflex exhibits a brighter tint, showing an increase of the arterial element in the chorioidal circulation. (6) Occasionally the retinal veins are observed to pulsate. (7) Reduction of ocular tension is common, even after a brief application of vibratory massage. Thus following vibration of the eyeball we observe reduced tension, improved vision, greater clearness of the media, enlarged retinal vessels; effects only explained by assuming that there has been an augmented flow of lymph and venous blood from the eye and an inflow of arterial blood with new formation of lymph and aqueous humor. (8) Applied directly to the eyeball, vibratory massage leaves a conjunctival hyperemia, lasting longer or shorter in proportion to the length of application. (9) Through the eyelids, vibratory massage usually reduces the hyperemia of the conjunctiva and so removes congestion in chronic episcleritis and allied conditions.

Every observation points to an equalization by the vibration of existing functional irregularities, in motor, sensitive, special and vasomotor nerve fibers, with their terminal apparatuses and central ganglia, resulting in the normal rhythmical action of each with all for their common object.

Applied, in appropriate cases, in a proper manner, patients uniformly speak of a feeling of comfort after they get accustomed to the strange sensations.

Usually I have resorted to vibratory massage only after other measures have failed, or in connection with such measures when unsatisfactory. Thus, some weeks since, in treating a young lady for a chronic thickening of the conjunctiva and subjacent tissue of both eyes covering the lateral recti muscles and reaching the sclerocorneal junction, two vascular ribbons failed to clear with ordinary measures, but a few treatments by vibratory massage relieved the deformity.

In cases of *early incipient cataract* with radiating striæ and surrounding opalescence, vibratory massage usually increases vision so that one or more additional letters of Snellen can be seen immediately after the treatment. Sometimes this is permanent, but usually transitory, as the conditions underlying the lens degeneration still remain. The explanation of this appears when we recall that the lens is nourished by lymph secreted by the ciliary and anterior chorioidal vessels, and enters the equator of the lens and after bathing its fibers escapes through the anterior capsule and with the aqueous escapes through the pectinate ligament and Schlemm's canal to the anterior ciliary veins; quickening this current, vibratory massage clears the media.

Illustrative of the value of vibratory massage in incipient cataract is the following: Mrs. S. M. applied for relief from failing vision and pain in eyes and forehead Feb. 27, 1907. She was a widow, aged 58, large and flabby, with muddy complexion. For many years she had supported her family by sewing, but for the last two had been unable to use her eyes much. Vision of right eye was twenty-seventieths and Jaeger No. 13; left eye twenty-fiftieths and Jaeger No. 13. Though she had half a dioptric of manifest hyperopic astigmatism, no glass improved her distant vision. Ophthalmoscopic examination showed striæ in both lenses, a hazy vitreous and small retinal vessels. Vibratory massage reduced the tension of both eyeballs; diminished the haziness of the vitreous; increased the size of the retinal vessels, and increased the vision in each eye to twenty-fortieths. In addition to the use of vibratory massage, attention was directed to rendering the secretions and excretions more normal, and hot water was locally applied to each eye, three times daily, for ten minutes. On Aug. 31, 1907, vision of each eye was twenty-thirtieths plus three letters, and with plus D. 3.00 S., Jaeger No. 1; four striæ had disappeared from the left lens and six from the right, leaving one stria in each lens mostly hidden behind the iris. Aside from these two striæ, the media of both eyes were quite clear. The pain in and about the eyes disappeared after the third treatment. Vibratory massage was used over and on both eyes, the nape of the neck and spine, three times weekly for the first month and less frequently later.

In cases of scleritis and episcleritis, vibratory massage has proved helpful, as in recent corneal opacities.

In case of recent chorioiditis, vibratory massage seemed to promote the clearing of the vitreous and hasten the absorption of the inflammatory deposits. The patient was a man, aged 56, healthy aside from a suspected syphilitic taint. For two weeks anterior to Nov. 12, 1906, he noticed a progressive failing in vision of right eye. On that date vision of the right eye was 20/100; considerable patch of Descemetitis in upper part of cornea, and a large patch of chorioiditis above the external to the macula. He was given full doses of potass. iodid internally; hot water directed locally three times daily for ten minutes, and vibratory massage once per week. On November 19 vision was 20/70; November 29 vision was 20/50; December 20 vision was 20/40; January 14 vision was 20/30; January 24 vision was 20/30 plus four letters.

After each treatment by vibratory massage, more letters could be read; sometimes one, other times two or more. This was generally retained till next treatment, when there was a farther increase. The Descemetitis vanished early, and the chorioiditis retracted its ders, though there remains a blind spot of considerable size.

In one case of optic nerve atrophy, vibratory massage was followed by an increase of vision in the right eye from twenty-seventieths to twenty-fiftieths plus one letter; in the left eye, from two-two-hundredths to four two hundredths. This improvement was present two and one-half months after treatment. Briefly, the case was a lady, 50 years old, who, twelve years previously, had suffered from nephritis, developing at the fourth month of pregnancy, and followed by miscarriage at the seventh month. Blindness in both eyes was complete for a long time, and she never became able to see more than the largest print with a magnifying glass. The visual fields were very restricted; pupils reacted but slightly to light; optic discs very pale (the left most); central vessels quite attenuated. The application of vibratory massage increased the size of the vessels perceptibly; slightly enlarged the visual fields; increased the visual acuteness as above stated.

In retrobulbar neuritis, from alcohol, tobacco, or both, vibratory massage has seemed to hasten recovery. The effect on the retinal vessels and general fundus could be seen readily with the ophthalmoscope and always showed an increased vascularity. The gain in vision depended on the stage of the disease. Sometimes a single treatment would increase vision by one, two or three letters; other times not at all. Sometimes the improvement would be permanent, other times not, according to the pathological conditions.

Among the functional disorders benefited by vibratory massage are cases of eyestrain, not fully relieved by the most perfect correction of both refractive defects and muscle unbalance. Many of these derive much comfort from vibratory massage to both eyeballs and nape of the neck. I recall the case of a steamboat engineer, many years a sufferer from awful headaches, in spite of the most perfect correction of a slight hyperopic astigmatism and a little esophoria. Vibratory massage applied as suggested relieved him, and an occasional treatment kept him quite comfortable.

Time forbids the presentation of detailed cases illustrating the field in which I have found vibratory massage useful, but I ask indulgence to a brief abstract of two cases as quite unique. My diagnosis was "anemia of optic nerve and retina." The effects of vibratory massage were prompt, striking and permanent.

Case 1.—On Jan. 21, 1904, Mr. W. F. E., aged 50, applied for relief from a persistent and rapid loss of vision, observed to begin about four months previous. Long a sufferer from piles, he had been successfully operated nine months before. He was a bookkeeper, with the best of habits, except he smoked from six to eight cigars daily. Because of chronic indigestion, he had been under the care of a stomach specialist. He said that he had been treated by several excellent general practitioners and ophthalmologists, besides his surgeons and stomach doctors. Early in October, 1903, he could see perfectly well to read or work as he desired. But soon after, he noticed failing vision, until at date of his visit to me vision was 18/200 right eye, 6/200 left eye, increased by glasses to 20/200 and 18/200. The glasses worn were cylinders minus one and one-half diopters against the rule, with minus one diopter sphere. No increase of vision could be had with any other combination.

Visual fields were limited, though color fields were normal over the visual area. Tension was normal, as were the movements of the iris and reflexes. The media was clear, but the fundus was characterized by a deficient blood supply, matching the thread-like veins and arteries; the discs were uniformly pale.

As no organic disease could be made out and the retina was clearly suffering from improper nourishment, vibratory massage seemed indicated, especially as I had already seen that it increased the retinal circulation. This was accordingly applied, for three minutes, to each eye, over the closed lids, reaching every part of the eyeball possible by extreme rotation in different directions. He said that he saw much clearer and his vision rose to 20/200 Snellen each eye immediately after the treatment. The ophthalmoscope showed an increased size of the retinal vessels throughout, and the entire area looked brighter.

In the hope of maintaining this, he was directed to locally apply hot water for ten minutes every four hours to each eye. He was also directed to take a glass of koumyss four times daily; outdoor exercise was prescribed and such other mode of life as would improve his general condition.

Daily thereafter vibratory massage was given, in varying strength, during longer or shorter periods, till vision became normal, when it was dropped to alternate days, until May 16 he was discharged with vision twenty-twentieths, Jaeger No. 1, able to read for hours without weariness. At the fourth treatment I noted that his vision was 20/70 right eye and 20/100 left eye. As seen by the ophthalmoscope daily, the vascularity of the fundus generally improved till it became normal at time of discharge.

A peculiarity of his improvement was the great difficulty in making an advance of a single letter one day and perfect ease in seeing the same letter on the following day or two.

He was restricted to three cigars daily, all smoked out of doors. At no time could retrobulbar neuritis be made out in accord with the judgment of the excellent eye men who saw him before his visit to me.

So far as a positive diagnosis could be made, the retina was starved by malnutrition, following a combination of rectal disease, chronic indigestion, mental worry and close use of his eyes. It seemed probable that irreparable organic disease was near, from starvation of the retina, had it not been averted by the means indicated. Now, nearly four years later, Mr. E. reports perfect vision, both near and far, with glasses, and ability to continue with ease close application to his books. There has been no failure in vision during these years, so that the restoration of vision was permanent.

Case 2.—Mrs. O. F. L., aged 35, on April 3, 1907, sought relief from almost complete loss of vision in her right eye, first observed three days previous. She was unable to distinguish a flame in any portion of the visual field; with difficulty she counted her fingers at one foot, and no lens improved vision. Ophthalmoscopic examination showed the media clear, the central retinal veins large and arteries mere threads. Vibratory massage was applied to the ball for three months, when vision became 1/200. This was repeated daily, and to it added the local use of hot water and the internal administration of increasing doses of iodid of potass. On April 6 vision was 3/200; April 8, 10/200; April 13, 20/70; April 15, 20/50; May 2, 20/30. On May 13 she was able to distinguish flame in any direction. As vision improved, the retinal arteries increased in size till they became normal. When last seen, on June 15, 1907, the retinal arteries were alike in each eye and vision 20/30, Jaeger No. 1. This was increased by correcting the hyperopic astigmatism with hyperopia to 20/20 in each eye, thus O. D. plus DI. C. ax 90 deg. plus DO. 25 S. — O. S. plus DO. 50 C. ax. 70° DO. 75 S.

When first seen, vision of left eye was 20/40, Jaeger No. 1. At last visit it was 20/30, increased to 20/20 by proper glass.

Time does not admit of reference to other classes of cases in which more or less satisfactory results were attained.

Vibratory massage was introduced to the profession by Maklakow in 1893. He employed Edison's electric pen, the point being covered by an ivory ball, vibrating about 9,000 per minute. He found intraocular tension reduced even in glaucomatous eyes; helpful results were secured in parenchymatous keratitis and episcleritis.

Since others have reported good results in maculæ, follicular and granular conjunctivitis, iridocyclitis, traumatic cataract and hypopion keratitis, Sneguirew, by experiment, showed that diffusion of fluorescin from the conjunctival sac towards the anterior chamber was markedly increased by vibratory massage while the tension was diminished. He explained this by saying that the vibratory massage quickened the lymph circulation in the cornea and conjunctiva.

DISCUSSION.

Dr. Fridenberg, New York:— I was much interested in the observations as to the clearing up of the lens. We know that some of these methods for cataract depend on massage, direct or indirect, over the lens: and it is a question whether we should apply this form until we know better how pressure acts on lenticular opacities. Of course there are opacities which clear slightly subsequently—sometimes permanently; more often the changes are but temporary. I think the general experience is that much massage does tend to cause the maturation of the cataract. The old fashioned massage, without vibration, I find most effective in treating black eye, with the fingers—a prolonged massage of the lids. Scleritis disappears more rapidly under dionin and massage than under any other method at our disposal.

Dr. Green, Jr., St. Louis, Mo.:—The observations of Hinshelwood in connection with the use of dionin in eyes not relieved by the correction of refractive errors seems to me interesting in this connection. He noted that weak solutions of dionin were beneficial, especially in neurasthenic individuals in whom the most painstaking correction of refractive and muscular errors had failed to abolish asthenopic symptoms. May there not be a certain analogy between the efficacy of dionin and the efficacy of vibratory massage? I would ask Dr. Connor, in closing, to particularize as to the instrument he uses and the duration and manner of its application.

Dr. Connor (closing discussion):—Massage depends much on the fitness of the instrument for work of vibratory massage on the eyes. The one I found most satisfactory was the Wrappler No. 2, mechanical and vibratory apparatus. With this, one has in perfect control the frequency, length and force of vibrations. Its construction admits of easy application to any part of the eyeball. The small acorn-shaped tip of hard rubber was used most frequently. I never observed any pain or discomfort to follow the use of vibratory massage. Vibratory massage is a different thing than pressure on the eyeball used for maturing a cataract, which is intended to destroy the cells of the anterior capsule of the lens, admitting aqueous thereto. This vibratory massage has reduced the striæ and so removed incipient cataracts in cases reported and a number of cases not reported here. A certain case of mechanical vibrations in and about the eye improve other morbid conditions and so help in cases that have heretofore seemed impossible of improvement. Vibratory massage elsewhere will do good, as has been proven; I am sure we can use it to advantage in some intractable cases.

MAGNETIC AND NON-MAGNETIC PROPERTIES OF IRON ALLOYS.

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During the past few years the increasing industrial use of the alloys of steel has resulted in marked changes and improvements in the quality of steel manufactured. The effect of these various alloys, as well as the percentage of carbon and whether annealed or unannealed, on the physical condition of steel, particularly its magnetic property, is of considerable importance to the ophthalmologist, especially those in the vicinity of large iron and steel industrial centers, on account of the possibility of injury from splinters of these newer steels as they are being used more every day in the construction of improved machinery and tools.

Although familiar in a general way, through a knowledge of metallurgy, with the magnetic properties of the different alloys of iron, my immediate attention to the importance of the subject was directed by the following case:

G. C., aged 21, consulted me on Nov. 24, 1905, with an injury of the right eye received while working on a dynamo bed-plate. The wound was a large one, ten millimeters long, and extended from the nasal side of the sclera into three millimeters of the cornea. The lens was ruptured, so that a view of the fundus could not be obtained. Under general anesthesia, attempts to extract the metal by the giant magnet failed. With such an extensive lesion, and believing that either the metal had gone through the eye or was embedded in the eye opposite the wound, enucleation was done. The globe was intact and the metal found in the bottom of the vitreous chamber. As I was at a loss to account for the negative reaction from so large a piece of metal, measuring 7 by 4 millimeters, and weighing 2¼ grams, I placed the fragment in the magnetic field, and, to my surprise, it was not attracted by the magnet. Upon inquiry, I was told that the bed-plate in question contained a high percentage of manganese.

The subject, which is of importance both from an ophthalmological as well as a scientific standpoint, will be considered under two headings: (1) Those alloys of iron which are non-magnetic or nearly so even in strong magnetic fields; (2) those alloys of iron which are more magnetic than the purest commercial iron, within a certain range of the magnetic field.

ALLOYS OF IRON WHOLLY OR NEARLY NON-MAGNETIC.

Manganese.—The alloying of iron with varying percentages of manganese and the effect it produces in the destruction and alteration of its susceptibility to magnetism is interesting and surprising. When manganese in an alloy of iron reaches 13 per cent., its magnetic character is destroyed, and steel thus produced is as inert to magnetism as zinc or stone. The effect of composite alloys of iron is even more striking. Nickel steel in itself is extremely magnetic; even an alloy containing 15 or 25 per cent. of nickel is fairly magnetic, but add 5 per cent. of manganese to any of these high percentage nickel steels and they become non-magnetic, although a 5 per cent. manganese steel is fairly magnetic, but of no commercial use, as steel containing 2.5 to 7 per cent. of manganese is brittle and comparatively worthless, but when the amount exceeds 7 per cent., great strength and toughness are obtained. Small quantities, 0.4 to 0.5 per cent. of manganese, are highly beneficial to steel and the very best steel contains a little. An 8 per cent. manganese steel has considerable intensity of magnetization, but add 2½ per cent. of nickel and it becomes non-magnetic, although a 2½ per cent. nickel steel is almost as magnetic in low fields as pure iron. This phenomenon is produced with other metals alloyed with manganese steel. A curious fact is observed when copper or chromium is added to an almost non-magnetic nickel-manganese steel. The composite alloy increases the magnetic perceptibility probably on account of the softening effect produced by the added metal. Another important point in the magnetization of iron alloys must not be lost sight of, and that is a body which in the annealed state is feebly magnetic in a certain magnetic field becomes non-magnetic in the same field when unannealed. As magnetization involves a molecular change in magnetic bodies, whatever affects the state of molecular aggregation in these bodies affects the magnetic susceptibility. An increase or decrease of mechanical hardness means an increase or decrease of the power of resisting molecular change, and hence magnetic hardness or magnetic softness corresponds to mechanical hardness or softness of iron and its alloys. It is thus easy to understand the conversion of a magnetic into a non-magnetic body by alloying the former with certain metals which increase the mechanical hardness of the alloy.

Excess of carbon in high manganese steels softens them and increases their magnetic susceptibility, so that high carbon steels having a percentage of from 13 to 15 per cent. manganese are slightly magnetic in powerful fields. With low carbon and the same percentage of manganese the steel is practically non-magnetic.

The great commercial and practical importance of manganese steel, on account of its hardness and toughness, is seen in its application in the construction of machinery and tools, as in rock-crushers, railroad crossings, frogs, car-wheels, safes and, on account of its low magnetic susceptibility, in bed-plates of dynamos.

ALLOYS OF IRON MORE MAGNETIC THAN PURE IRON.

The magnetic susceptibility of the best and purest commercial iron is increased in low fields by the addition of nickel, silicon and aluminum.

Nickel.—Owing to its cheapeness, nickel-steel is more extensively used than any of the other hardening alloys. When nickel does not exceed 3 per cent. the alloy is nearly as good magnetically as very soft iron, and when the magnetic field is very low the permeability of a 30 per cent. nickel-iron alloy exceeds the best and softest iron.

In armor-plate, axles, railroad forgings and rails, the percentage of nickel averages 3 to 3.5 per cent. In marine and locomotive boiler tubes, the percentage of nickel varies from 25 to 30 per cent.

Chromium.—Chromium has a much less powerful effect in destroying the magnetic properties of iron than manganese, for iron containing 65 per cent. of chromium is still attracted by a magnet. It is used either alone or with nickel or tungsten in burglar-proof safes, tools and armor-piercing projectiles. Commercial steel contains 3 to 5 per cent. of chromium.

Tungsten.—Tungsten united with copper produces some valuable alloys. Steel containing tungsten is highly valuable for cutting tools. Tungsten steel possesses remarkable magnetic properties, as used commercially in percentages of from 3 to 10 per cent.

Two and a half per cent. of *silicon* or *aluminum* alloyed with iron increases the magnetic permeability so that the magnetism is better than the purest Swedish charcoal iron. When aluminum in ferro-alloys exceeds 17 per cent., the alloy becomes non-magnetic.

The fact that special steels differ widely in their behavior when placed in a magnetic field ought to be borne in mind, although all of them bear only slightly or indirectly on the question of the possibility of removal from the eye.

DISCUSSION.

DR. ALT, St. Louis, Mo.:—I suppose we have all had difficulty in extracting pieces of steel from the eye, when the attraction was altogether impossible. The usual explanation has been that the piece was encapsulated in lymph and that in consequence the magnet could not pull it out. I always thought that a somewhat doubtful explanation of non success, and I wonder now if not in most of these cases the fact was the steel was non-magnetic. I think the knowledge which Dr. Frank has imparted to us is of great value. At least it gives us a satisfactory explanation if we do not succeed in getting a piece of steel from the eye by means of the magnet, although we are perfectly satisfied that it is there.

Dr. Connor, Detroit, Mich.:—Dr. Frank's studies emphasize the fact that we should not decide a foreign body is not in the injured eye unless we take an *x*-ray. Some men think if the magnet produces no reaction it is sufficient evidence that there is nothing in the eyeball. In the light of this paper it would be fair to assume that even when we do not get a reaction with the magnet the steel may still be there, though of that quality which does not react to the magnet.

Dr. Black, Milwaukee, Wis.:—Dr. Frank has given us some very helpful suggestions, and I wish that he would give us an idea in what industries we will find the steel used to be magnetic and which non-magnetic.

Dr. Green, Jr., St. Louis, Mo.:—One lesson enforced by Dr. Frank's paper is that henceforth we should take very accurate histories of cases with special reference to the probable character of the steel in the eye. If this information can not be obtained from the patient himself, we should make inquiry of some responsible person connected with the foundry as to the character of the steel before venturing on any operative procedure.

Dr. Suker, Chicago:—While Dr. Frank was working on his paper I had a case which would about fit his description. A man employed in the steel mills had a small piece of steel embedded in the eye near the ciliary bodies. The magnet failed to give any reaction whatsoever and the piece of steel was so embedded that the *x*-ray was practically negative. I advised enucleation. While I was making microscopic sections the piece of steel was found in said location. Upon holding the section up to the magnet no attraction whatever ensued. The piece of steel was so firmly embedded that I could not get it out without destroying the section. It must have been one of those high alloys of which Dr. Frank has spoken.

Dr. Frank, Chicago (closing discussion):—Replying to Dr. Black's question as to what institutions used the different alloys: The Taylor Iron Works of High Bridge, N. J., manufacture the largest amount of high manganese steel in the country. All the rock crushers are made by them, and those large deep shovels with long teeth, safe doors, car wheels, steel street cars and railroad crossings. Most factories that use any steel have a record of where they get their steel and the percentage of alloys in it. They will tell you if you have occasion to call on them. They can give you the exact chemical constituents of the different products. As Dr. Alt says, there must be a great many cases reporting failure of magent extraction, where the foreign body was either non-magnetic or very weakly so. I did not catch the meaning of Dr. Connor's remark about the *x*-ray, but in my case the wound was so large there was no doubt. But when in doubt I have an *x*-ray taken.

METHODS OF ILLUMINATING TEST-TYPE CHARTS WITH ARTIFICIAL LIGHT.

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Solar light as diffused by an overcast sky gives ideal illumination. However, the variations produced by the weather, the time of day, the inaccessibility of daylight to rooms where vision testing is done, has of necessity enforced the adoption of artificial means to illuminate objects used for measuring visual acuity.

Professor Basquin,[1] of Chicago, has determined that the variation in light from the clear sky was from 1,050 candles per square foot in August to 140 candles in December of the same year.

The days were divided into five classes, No. 1 stormy, No. 2 overcast, No. 3 more than half cloudy, No. 4 more than half clear, No. 5 clear. The mean monthly brightness of the sky at 12:30 (noon) for two years, expressed in candles per square foot, was: Class 1, 310; class 2, 960; class 3, 750; class 4, 590; class 5. 460. The strongest illumination was on overcast or cloudy days, there being better diffusion.

The mean of the monthly readings, without regard to class of sky, varied from 2,200 candles per square foot in June to 270 in December. The mean monthly readings taken at 4:30 p. m., without regard to class of sky, varied from 520 candles per square foot in August to 5 in December.

It will be seen that this great variation makes daylight illumination impractical for the purpose of testing visual acuity. Some means of artificial illumination must take its place. Petroleum, illuminating gas, acetylene gas and electricity place at our disposal an unvarying, constant means with which to accomplish this purpose.

Of these electricity is the most convenient for our use, as practically every city of sufficient size to support an ophthalmic surgeon is lighted by electricity. Petroleum, illuminating gas and acetylene gas are equally efficient from the standpoint of candle power derived, but are less satisfactory to manipulate.

As this paper refers only to methods of illumination, the amount of light necessary for proper illumination of test charts will not be referred to, except to state that it has been determined that with a white surface having a relatively high coefficient of reflection, with an illumination of one to two-foot candles, the eye is working so near its normal sensibility that further increase of the illumination is of relatively small value.[2] The committee appointed by the ophthalmic section of the American Medical Association to determine upon a standard of illumination for test-type charts will submit a full report upon this subject at the Chicago meeting next year.

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[1] Basquin, O. H.: "Daylight Illumination," Illum. Eng., December, 1906.

Artificial illumination may be used in two ways, either reflected or transmitted. There is considerable difference in opinion throughout the United States as to the efficiency of these two methods, and to obtain an expression of opinion from the leading ophthalmologists, the following questions were sent out:

"With the use of artificial light for illuminating test-type charts, which do you consider the most efficient, reflected or transmitted light?

"Why?

"Which gives the least discomfort to the examined persons' eyes, especially when testing under a cycloplegic?"

Sixty-two replies were received and were classified as follows: Eighteen preferred reflected light; 9 preferred transmitted light; 5 used daylight only; 3 found practically no difference; 25 had had no experience with transmitted light; 1 had no opinion to offer; 1 question as asked not answered.

The following replies contain some suggestion or reasons for the opinion expressed:

Dr. Alvin A. Hubbell: "I have never used transmitted light. Reflected light is adequate and agreeable, providing the cardboards are not glazed and shiny."

Dr. A. D. McConachie: "I use reflected light. Never have used transmitted. However, can conceive that the transmitted might be better when confined to a small area, and single row of test-type at a time."

Dr. Kaspar Pischell: "I have always used reflected light. It is nearer the actual conditions under which we see."

Dr. W. R. Parker: "Have never used transmitted light for illuminating test-type charts. Reflected light has been satisfactory, but I think transmitted light might be more restful."

Dr. C. A. Thigpen: "I find that reflected light (artificial) is more trying to the eyes under a cycloplegic than the natural solar light."

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[2] Bell, Louis: "Some Physiological Factors in Illumination and Photometry," Illum. Eng., June, 1906.

Dr. Myles Standish: "Generally speaking, the intensity of illumination of test card should not be overpowering in comparison with that of surrounding objects."

Dr. Leartus Conner: "All depends upon the amount of light entering the eye; generally as used by me the reflected light is best. But other arrangements might reverse the order."

Dr. Samuel Theobald: "Reflected; we commonly 'see things' by reflected light; have had no experience with transmitted light."

Dr. Bert Ellis: "Reflected light onto white letters on a black background."

Dr. Edward Jackson: "Reflected light, because a greater variety of test cards can be employed and there is less radiation." White letters on a black ground give least discomfort to examined persons' eyes.

Dr. John E. Weeks: "The irradiation incident to the use of transmitted light seems to be the chief trouble. This appears to be more marked (the annoyance) when the pupil is dilated than when the pupil is small."

Dr. Henry Dickson Bruns: "Reflected; it more nearly simulates the normal conditions."

Dr. Geo. F. Suker: "Reflected light, patient to sit in very dark room, card dull finish, reflection limited if possible on to card alone."

Dr. Chas. H. Williams: "Reflected, because the intensity of the illumination can be controlled and measured more accurately, and because it is in more general use and the results can be better compared. I have the opal glass test-type, but have not used them enough in comparison with the paper to make a proper answer to this question (No. 3). The yellow paper, however, seems to be more agreeable than the white."

Dr. F. M. Chisholm: "Reflected, softer light and for test purposes more like daylight, as uniform and less artificial than transmitted light types, owing to sharp contrasts from field of illumination and its surroundings in the latter."

Dr. J. H. Claiborne: "Reflected light. Have no other. Never heard any complaint, though have, of course, noticed lack of good definition owing to spherical aberration under A or H."

Dr. J. W. Chamberlain: "Reflected, gives perfect illumination and is easily managed."

Dr. Frank C. Todd: "Reflected, light is more even and easier on the eyes."

Dr. D. T. Vail: "Reflected, since we see everything by reflected light. I like a mixture of electric and daylight, as daylight grows dim the electric grows brighter, making the illumination always uniform."

Dr. J. E. Colburn: "Reflected light. Better results, especially in the astigmatism tests. The transmitted light of equal intensity causes pain and blurring, with longer and more frequent rest."

Dr. A. O. Pfingst: "Reflected, it gives better definition of letters; in other words, the contrast between black and white is sharper."

Dr. J. A. White: "I use as a rule reflected light. I use transmitted light sometimes, but it does not give as brilliant an illumination as the reflected lights.'

Dr. Geo. M. Gould says: "Your question is meaningless to me and according to my use of test-types. The tiring of the eyes is bad both ways with white background and black letters. I do not follow this ophthalmologically absurd method. I use cards put up on the physiologically correct principle of black background and white letters."

Dr. H. V. Wurdemann: "Transmitted electric light through porcelain plates, always of uniform intensity; reflected light gives confusing reflections, on porcelain plates—paper charts fade and the standard thus varies."

Dr. Melville Black uses transmitted light with revolving cabinet and says: "Sometimes when the electricity is turned off for a while I use reflected light or daylight, and invariably the patient complains or rather expresses relief when the electricity comes on again and the blinds are pulled down."

Dr. C. H. Beard: "Transmitted light, chiefly because of the absence of the many surface reflections which are objectionable in other methods."

Dr. Linn Emerson: "Transmitted; more illumination of chart. No reflection from shiny surface of card."

Dr. F. H. Verhoeff: "Theoretically it seems to me that transmitted light should be more efficient because it would obviate luster. However, I have never used it because I have not seen a practical method of doing so."

Dr. Casey A. Wood: "Transmitted light, because it seems to be less irritating to the eyes both of the examiner and the examined."

Dr. F. T. Rogers, speaking of his cabinet, says: "The illumination, to my mind, is the most satisfactory that I have ever had and, inasmuch as I illuminate one line at a time, there is not the large white space to cause fatigue to the patient's eyes. They can look at it steadily without discomfort, whereas if all the lights are illuminated at once there is some annoyance."

E. L. Elliott, editor of the *Illuminating Engineer*, in reply to a letter relative to the illumination of test-charts, says: "As it is impossible to secure actual normal daylight illumination in the majority of cases in which test-type charts must be used, an artificial light should be taken as a standard. Almost all objects are seen by reflected light. Luminous bodies form an almost infinitesimal portion of objects viewed. Translucent test-cards which are practically luminous bodies should therefore be wholly discarded. In selecting a standard light source for illuminating test-charts that one should be chosen which produces a light nearest in quality to normal daylight. Oil and gas flames and the incandescent electric lamps all fall far short of meeting this requirement, the light which they produce being orange yellow, that is, has far less rays in the blue and violet than normal sunlight. Fortunately there is a light source available at the present time which approximates daylight to a remarkable degree. This is the acetylene flame."

The following reasons were given in support of reflected light:

1. "We see everything by reflected light."
2. "Nearer actual conditions under which we see."
3. "Less irradiation."
4. "Softer, light more like daylight."
5. "More even and easier on the eyes."
6. "Transmitted light of equal intensity causes pain and blurring.
7. "Contrast between black and white sharper."
8. "Gives a more brilliant light."
9. "The intensity may be controlled and measured more accurately."
10. "In more general use and records can be better compared."
11. "A greater variety of test-cards may be used."
12. "Transluminous test-charts which are practically luminous bodies should therefore be wholly discarded."

In reply to the above argument it may be stated that we *do not* "see everything by reflected light, nor is illumination by reflected light nearer actual conditions under which we see." In looking at a picture, reading a book, looking at objects lying upon the floor or upon a table we see by reflected light.

In looking at a landscape or a marine view or, for instance, in determining the position of a railway signal (semaphore) it is not reflected light that enables us to make out the contour of a building, tree, ship or semaphore, but the light that comes from behind the object viewed; the blotting out of a portion of the illuminated background makes the object visible.

Irradiation is the "phenomenon of the apparent enlargement of an object strongly illuminated when seen against a dark ground" (Century Dictionary). A white square surrounded by black appears larger and less sharply outlined than a black square surrounded by white, under the same intensity of illumination. "The effect of irradiation is most manifest when the dark portion of the field of vision over which the irradiation takes place has considerable breadth."[3]

There is certainly more irradiation from a card illuminated by reflected light than from perfectly diffused transmitted light, otherwise the diffused daylight coming through our windows would cause more annoyance than the light reflected through the same window from some nearby building.

Light as it emerges from translucent charts properly prepared is much softer, more like daylight and much easier on the eyes because it is perfectly diffused. The orange yellow of the light source is to some extent absorbed or filtered out by the porcelain.

There is, however, a yellowish tinge to the transmitted light, and while it has been determined that the portion of solar spectrum from D to E produces the greatest bleaching of the visual purple, diminishing with diminishing wave length (red being least active, yellow slight, and yellow green greatest), there is no doubt as to the truth of Dr. Williams' statement that "yellow paper seems to be more agreeable than the white."

The *Illuminating Engineer*,[4] in referring to "color of light as a factor in efficiency," says: "It is an easy enough matter to produce carbons which will give practically daylight values, but the efficiency is thereby cut in half. This is a physiological necessity from which there is absolutely no escape. For every purpose, therefore, in which distinctions of color are not of prime importance, light of a distinctly yellowish color is likely to be the dominant artificial light for a long time to come. That a light of this color produces an effect that is agreeable to the eyes is a further point in its favor."

Tschering says:[5] "Yellow occupies a special position among the colors and is that one of the spectral colors which, to the eye, seems to offer most resemblance to white."

Wm. A. Ryan says:[6] "Yellow light appearing to be easier on the eyes than white light is a matter of intrinsic brilliancy rather than color."

"The importance of subduing the intrinsic brilliancy of all light sources that come into the range of vision can not be overestimated.

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[3] Amer. Text-Book of Physiology.

[4] "Color of Light as a Factor in Efficiency," Illum., Eng., June, 1906.

[5] Tschering: Physiologic Optics.

[6] Ryan, W. D'A.: In Discussion, Illum. Eng., September, 1906.

. . . A frosted lamp or diffusing globe, while actually reducing the amount of light given out, increases the illuminating effect; that is, enables the eye more readily and with greater ease to see the illuminated objects."[7]

The difference between the effect of the intrinsic brilliancy of an area illuminated by reflected and transmitted light upon a sensitized plate is shown in the photographs. Both were illuminated by one 16 candle-power lamp with the plate at a distance of forty-two inches.

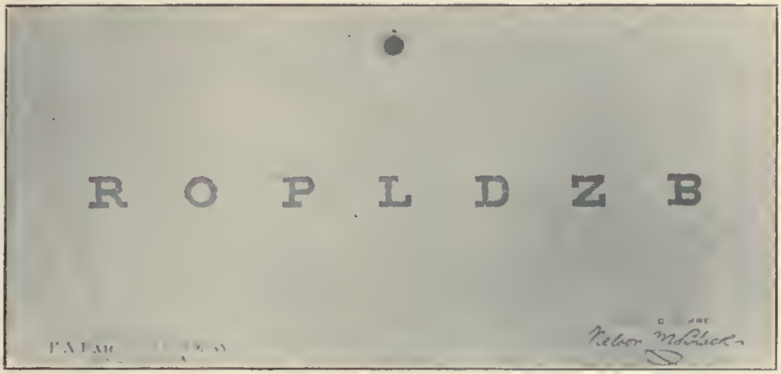

Fig. A.—Illustrates the effect of reflected light on a sensitized photographic plate exposed two minutes at forty-two inches distance. Illumination one 16 c. p. lamp at nine inches. Letters begin to show effect of over exposure and are blurred.

Figure A, the chart illuminated with reflected light, was exposed two minutes.

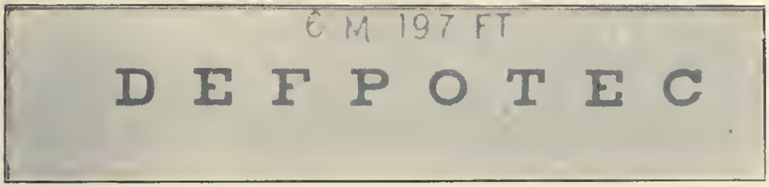

Fig. B.—Illustrates the effect of perfectly diffused transmitted light on a sensitized photographic plate exposed four minutes at forty-two inches distance. Illumination, one 16 c. p. lamp at two inches. Letters are sharp, clear cut, and distinct.

Figure B, illuminated by transmitted light, was exposed four minutes. The plates were developed together, in the same solution and for the same length of time. Figure A shows beginning haziness due to overexposure, while Figure B is clear and distinct.

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7. Elliott, E. L.: "Plain Talks on Illum. Engineering," Illum., Eng., January, 1907.

The intensity of illumination with transilluminated charts need not be as great by a considerable amount to obtain the same visual acuity as with reflected light.

Testing with Williams' simplex photometer, I find I can distinguish the 6/vi letters at 6 M. with the photometer set at 3 magnitudes, illuminated with transmitted light from 10 c.p. lamp, and with reflected light from a 16 c.p. O'Brien straight filament lamp the letters are blurred beyond the 2 magnitude mark.

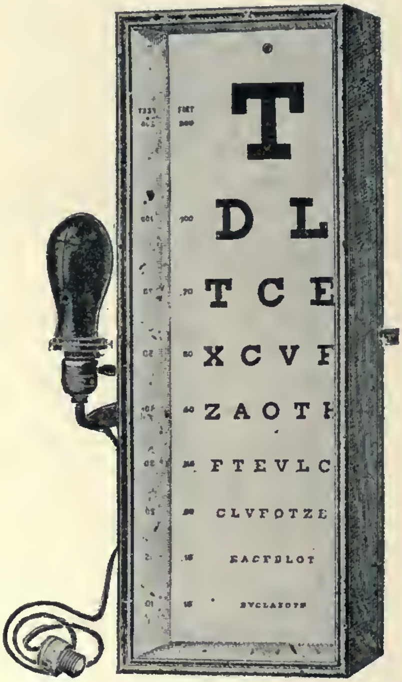

Fig. 1A.—Meyrowitz' illuminating test-type cabinet.

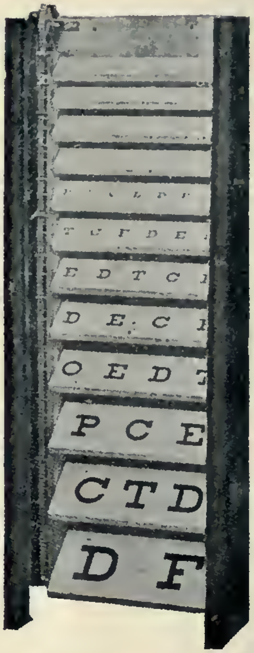

Fig. 1B.—Williams' test-type cabinet

The result of my personal experience is directly opposite to that of Dr. Colburn, i. e., that "transmitted light of equal intensity (to reflected light) causes pain and blurring." It is softer and much more agreeable to the eyes of the examined and the examiner.

Contrast between black and white is much sharper with transmitted light; compare charts.

Control of the intensity of the light is as easy with one method of illumination as the other, if electricity is the source. All that is necessary is to install a rheostat.

True, the illumination of test-charts by reflected light is in more general use, but I do not think, eliminating the question of intensity of illumination, that records of visual acuity will suffer by comparison if transmitted light is used. The variety of test-cards that may be used with transmitted is as unlimited as with reflected light.

That "transluminous test-charts are practically luminous bodies and therefore should be wholly discarded" is an argument that will not, as the old saying goes, "hold water."

Reflected light is necessary in order to appreciate the third dimension. Visual acuity as determined to-day is the measurement of the visual angle by means of characters of a definite size, upon a strongly contrasting background, which is in the same plane and is viewed from a distance. The length and breadth, or outline of the characters and their component parts, is all that is required for their recognition; the third dimension is not necessary for the purpose of testing visual acuity.

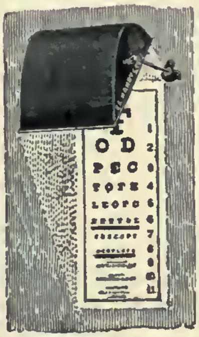

Fig. 2.—Frank's parabolic reflector for illuminating test-type.

Transmitted light for illuminating test-type charts is the best for the following reasons:

1. It is as near the actual conditions under which we work, in using the eyes for distant vision, as reflected light.

2. By this method there is no irradiation with black letters.

3. The light is softer upon a white ground and easier upon the eyes of the person examined and the examiner.

4. The contrast between black and white is sharper and the letters are more strongly outlined.

5. It gives a more perfectly diffused light free from confusing surface reflections and luster.

6. The intensity may be controlled perfectly by means of a rheostat, and the measurement of the intensity of the illumination is as easy as with reflected light, because the same means are employed (an illuminometer).

7. The variety of test-cards may be unlimited.

8. As large or small an area of the chart may be illuminated as may be wished for.

9. The letters being opaque, are seen perfectly black.

Just a few words concerning Dr. Geo. M. Gould's[8] "Cards put upon physiologically correct principle of black background and white letters:"

I maintain the condition is physiologically incorrect for the following reasons: 1. Dark characters upon a lighter background have been in use from time beyond recall and the human eye has been adapted to this condition. 2. The irradiation is marked. 3. The characters appear larger and are not sharply outlined. 4. Luminous points or small brightly illuminated area tire the eye much more than dark areas surrounded by brighter ones. 5. The production of disagreeable after-images. 6. Tscherning says, "We can not identify the examinations with the luminous point on a black ground with that made by means of a black point on a white ground."

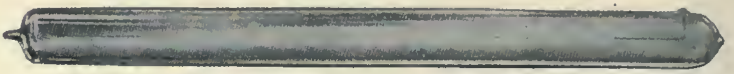

Fig. 3.—The O'Brien straight filament tube lamp.

Fig. 4.—The O'Brien lamp and reflector.

A comparison of the two charts, one black letters on a white ground, the other white letters on a black ground, both having the same illumination, will quickly decide which has the greater irradiation.

METHODS OF ILLUMINATION WITH REFLECTED LIGHT.

A large percentage of ophthalmic surgeons have their charts illuminated in almost "any old way." A card is stuck upon the wall with from one to a half-dozen lamps of various candle power either at the sides or top and bottom of the card; usually tin reflectors are used to direct the light upon the card, and very little attention is paid to the angle at which they reflect the light.

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[8] Gould, G. M.: "Test Cards with Black Background and White Letters," Ann. Ophth., January, 1897.

Figure one illustrates a method of illumination of which Meyrowitz, in his description of this cabinet, says: "Heretofore, unless a comparatively large number of lamps were used, even illumination of the card was not possible because of the limited area covered by a single lamp and shadows alternated with light areas. The four sides of the cabinet herewith presented contain mirror reflectors, which cause an even dispersion of the light from the single sixteen-candle-power lamp in all directions, so that the whole card is brilliantly and uniformly illuminated."

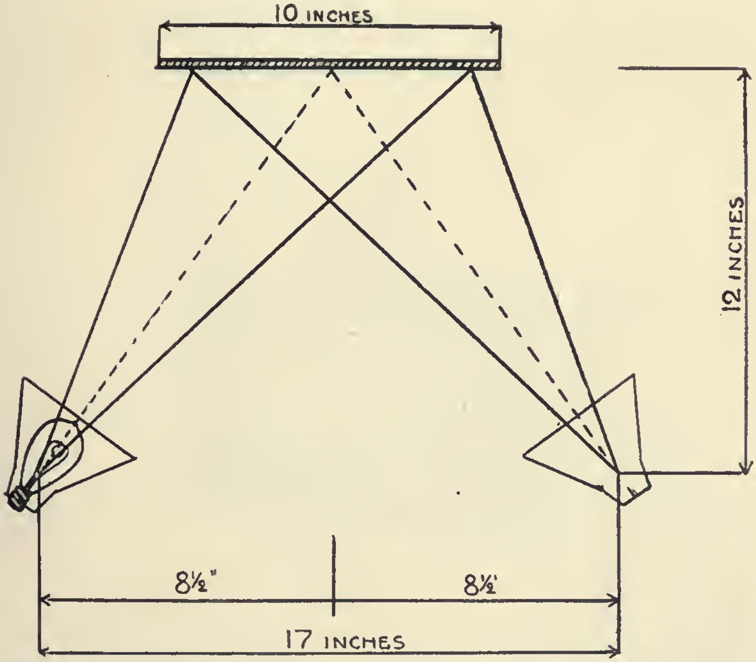

Fig. 5.—Scheme for lighting a card by reflected light, suggested by the Holophane Company.

Dr. Chas. H. Williams[9] says: "The most satisfactory results have been obtained by hanging the card of test-types in a cabinet painted a dull black, the opening being three feet wide by four feet high. On each side of this rectangular opening, right and left, is arranged a vertical column of small incandescent lamps of five candle-power each, eight on each side, made to burn four in series on a current of 110 volts.

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[9] Williams, C. H.: "A More Uniform Standard for the Illumination of Visual Test-type," Trans. Sec. Ophth. A. M. A., 1906.

"These lamps are arranged at equal intervals, the highest and the lowest being as near the top and bottom of the cabinet as possible, and are so placed that the lamps of one side come opposite the intervals between the lamps of the opposite side, thus making a more even distribution of the light. This vertical column of eight lamps on each side is protected by a blackened tin screen so that no light will pass directly from them to the observer, but so that the full amount of light will be thrown on the test-card. The lights are one foot in front of the plane of the test-types and about fourteen inches from the nearest edge of the card, and 'frosted' bulbs are used."

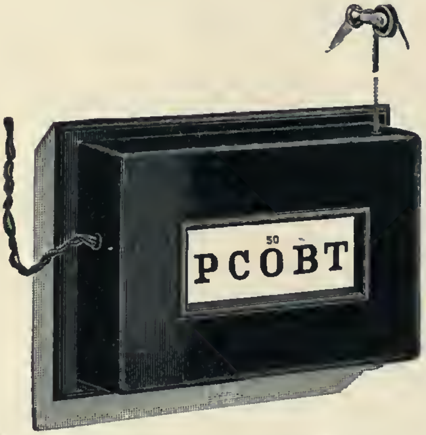

Fig. 6.—The Hardy 20th century testing cabinet.

Dr. Mortimer Frank says:[10] "The most common fault with the lighting of test-types is the improper care in the selection of the reflector and the position of the lamps." He has constructed a parabolic reflector which seems to meet the requirements perfectly, its only objection being the ungainly appearance of the apparatus, which I am sure the originator can improve upon (Fig. 2).

The O'Brien Electric Light Co. of Philadelphia have devised a lamp which, together with the reflector furnished, about meets all the requirements in the use of reflected light for illuminating test-cards. The lamp is of clear glass tubing, seven-eighths inch in diameter, with the filament running straight the entire length, giving a solid line of light twelve inches long. There being no sockets used, there are no shadows or dark spots (Fig. 3).

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[10] Frank, Mortimer: "A Parabolic Reflector for the Illumination of Test-types." Trans. Sec. Ophth. A. M. A., 1907.

The reflector is metal, lined with porcelain or opal glass, giving perfect diffusion of the light (Fig. 4).

The charts may be illuminated either by one lamp at the top and one at the bottom of the chart, or by one or two at the sides.

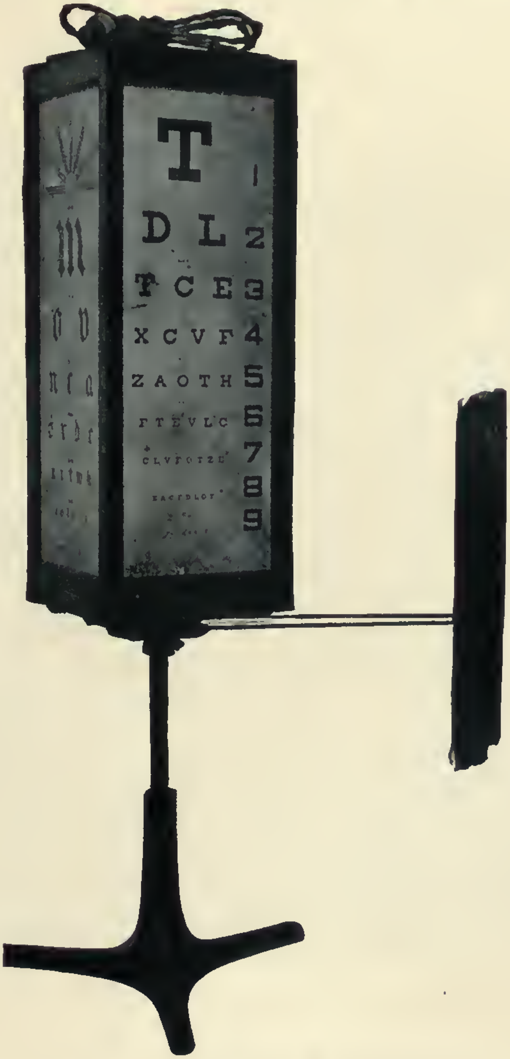

Fig. 7.—Black's luminous test-type cabinet.

I use one of these lamps and cards having a single line of type upon them, hanging the card on the wall, the lamp being suspended from adjustable brackets, which allows it to be brought within four inches of the wall or removed to a distance of nine inches, and also permits tilting of the reflector to any angle.

The engineering department of the Holophane Co. give the following scheme for illuminating a chart 10 by 25 inches: "By placing the reflectors 17 inches apart and 12 inches back from the placard and pointing in the direction shown in the sketch (Fig. 5) (50° and 125°) you will receive even illumination, and at the same time entirely hide the light from the person's eyes being examined. The reflector should be painted with aluminum paint on the inside and black or green on the outside. The number of lamps used, for best purposes, would be eight candle-power lamps, of which four should be placed on each side, up and down; six can serve the purpose, however."

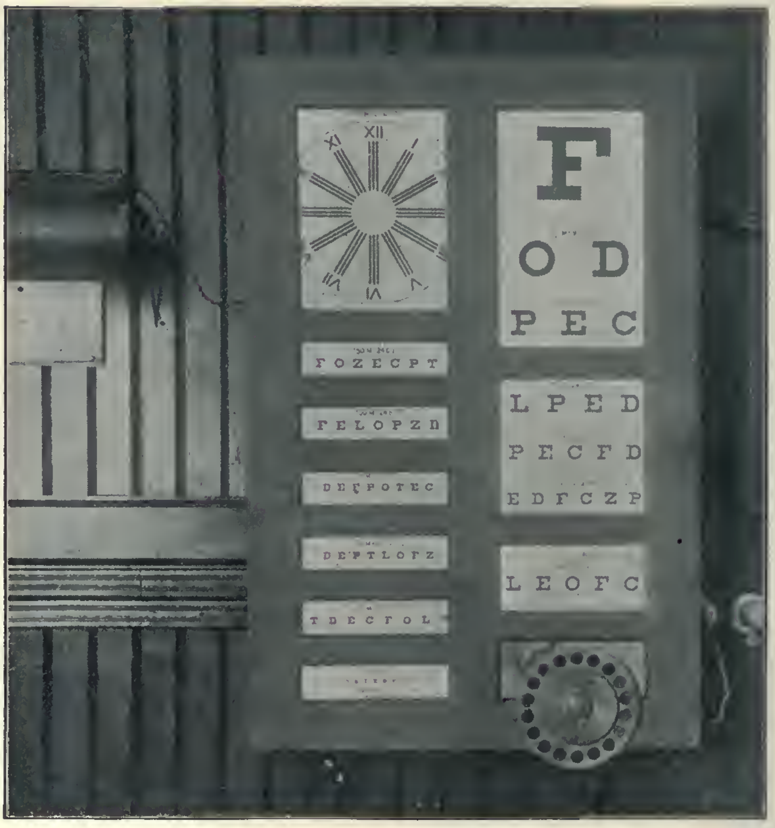

Fig. 8.—Transluminous test-type cabinet devised by Nelson M. Black, M.D., with astigmatic chart, light for muscle test and Williams' lantern for testing color vision.

TRANSMITTED LIGHT.

Hardy's Twentieth Century Testing Cabinet (Fig. 6) is one which is illuminated by transmitted light. The characters are printed upon paper, which by means of two rollers operated by a string allows one line of type to be seen at a time. This makes a good cabinet, but the paper becomes soiled and browned from the heat of the lamp and soon becomes inefficient.

The writer[7] has been using for the past five years a luminous test-type cabinet which has given very good results. It consists of a wooden frame, the four sides of which are mounted with porcelain transparent plates, on which the test-letters are engraved in dead black. The cabinet is illuminated by four incandescent lamps of 8 candle-power each, which give a very uniform illumination of apparently sufficient intensity. The four plates, having white glazed backs, reflect and refract the light in every direction so that practically none of the original illumination is lost, excepting what is absorbed in passing through the porcelain plate. The cabinet re-

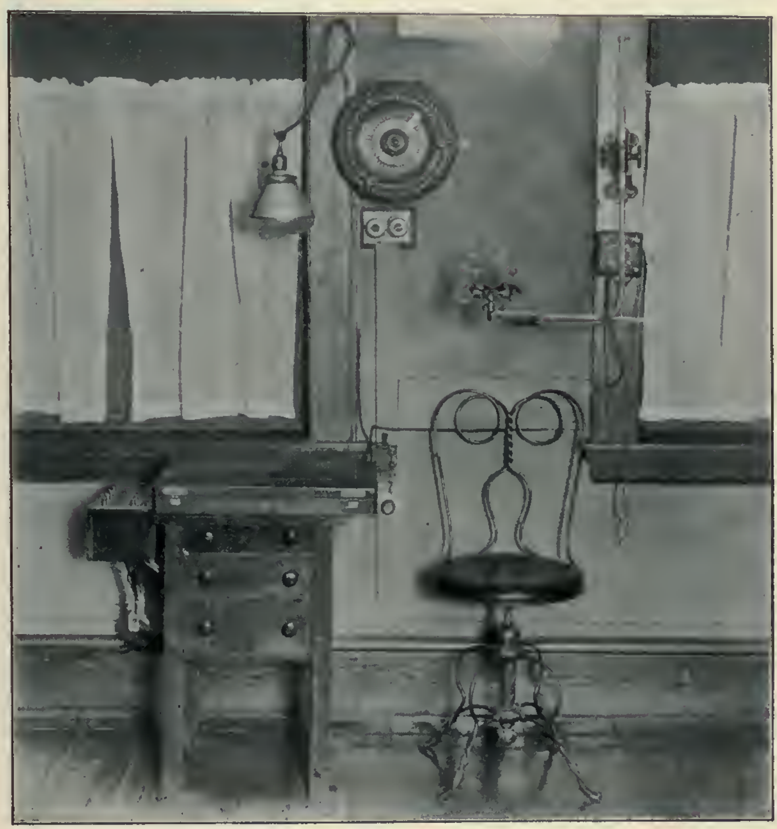

Fig. 9.—Showing refraction table, with switch board for operating different compartments of transluminous test-type cabinet and rheostat for controlling intensity of illumination.

volves on a central tube by means of cord and pulleys as shown in cut (Fig. 7). The objections to this cabinet are two: The surface illuminated is too great and has a tendency to tire the eyes, especially when examined under a cycloplegic, as does reflected light, although not to as great a degree. The number of lines of type exhibited at a time is confusing and it takes up a great deal of time unnecessarily to get the patient to attempt to read the line you wish.

The writer, in the following described cabinet, has attempted to overcome the objections (Fig. 8).

The size of cabinet outside is 21 by 32 by 7⅞ inches and is painted dead black. The left half is divided into seven compartments. The upper contains an astigmatic chart, the next two compartments contain letters for 6/vii½ vision, the next three have 6/vi letters and the last 6/v. The right side has four compartments. The upper contains letters for 6/lx, 6/xxx and 6/xx. The second compartment letters for 6/xv, 6/xii and 6/x vision, the next compartment 6/xii letters. The fourth compartment is fitted with an iris diaphragm for muscle testing and Williams' lantern for testing color vision. The inside is white enameled and the compartments divided by means of bright tin, which refract and reflect the light perfectly. The large compartments are lighted with two 10 c.p. incandescent lamps, the next smaller compartment with one 16 c.p. lamp and the small divisions with one 10 c.p. lamp each. The lamps are each turned on and off by a switchboard at

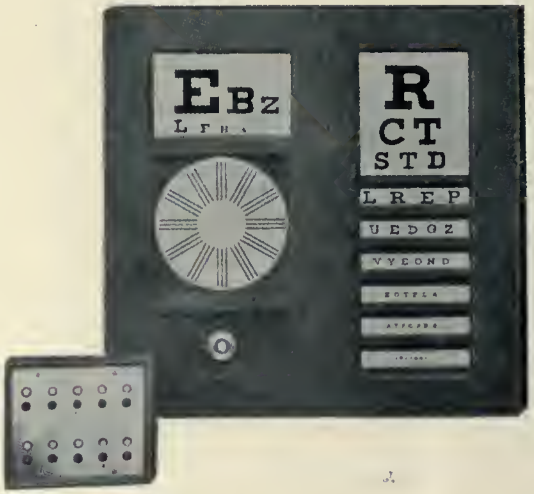

Fig. 10.—Rogers' luminous test-type cabinet.

the refraction table (Fig. 9), and the intensity is regulated by a rheostat so that the illumination may be controlled at will. Much to my sorrow, I learned as I had almost completed my cabinet that Dr. F. T. Rogers, of Providence, R. I., had preceded me by almost a year with the same idea.

Dr. Rogers' cabinet (Fig. 10) is 30 by 29 by 6½ inches in size. The left half is divided into three compartments; the upper,

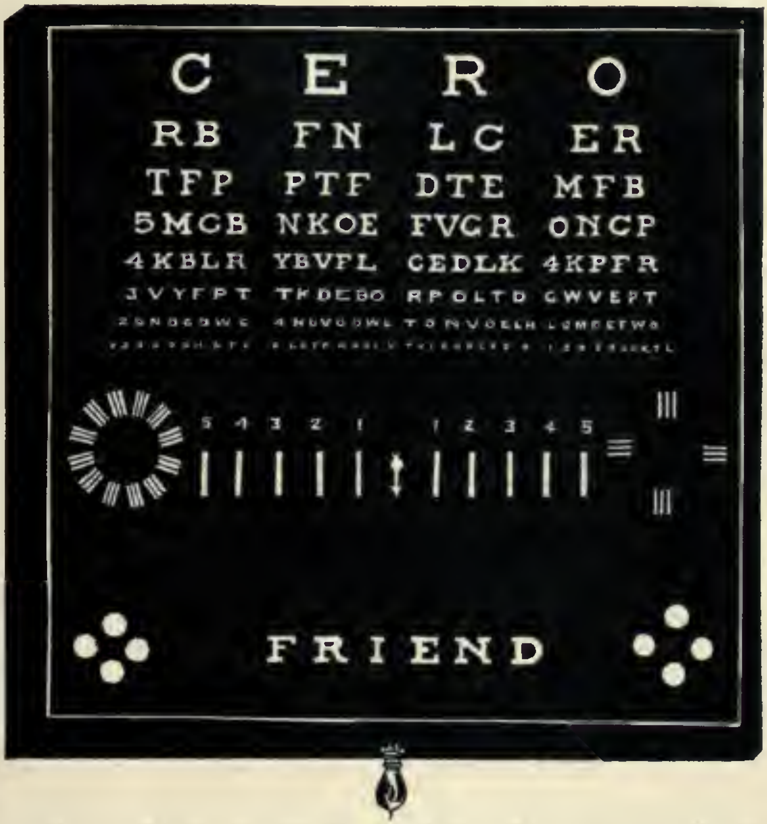

Fig. 11.—Welsh's luminous test-type cabinet. White letters on a black ground, 1/9 actual size. The numbers are one side white, the other side dark green. The circles are two green, one white, and one red. The word "friend," f, i, n is in green, r, e, d is in red.

being used for the primary test, has nine single letters decreasing in size, which enables the examiner to find what the acuity of vision is. Next below is an astigmatic chart, and at the bottom, behind an iris diaphragm, is a white light for muscle testing, also a slide with red and green glass for railroad signal tests. The right side is divided into seven compartments. The six smaller openings have duplicate lines of type which can be instantly dropped in or out of position as desired by the examiner. Each compartment is controlled by a switch at the refraction table.

Dr. Emmett Welch, of Grand Rapids, Mich., has devised a transluminous cabinet which he has used constantly for the last three years (Fig. 11). He describes it as follows:

"My room is a darkened room. The letters are made after Snellen test-type, actual size. The 'board' is plate glass painted black with white letters, the same having a frosted tint. All letters, numbers, circles and lines are frosted. The plate glass is 54 by 54 inches. Back of the same are electric lights, 8 candle-power. The glass rests in a frame composed of one side wall and window frame the other. Its depth is 10 inches. I manipulate the switch at twenty feet, illuminating all at once. This gives me, first, selection of letters for each eye singly and combined vision; second, astigmatic test; third; maddox rod test; fourth, to detect malingering, as you note the word 'friend.' Alternate letters spell two words and are painted in red and green, so by destroying the color by using alternately red

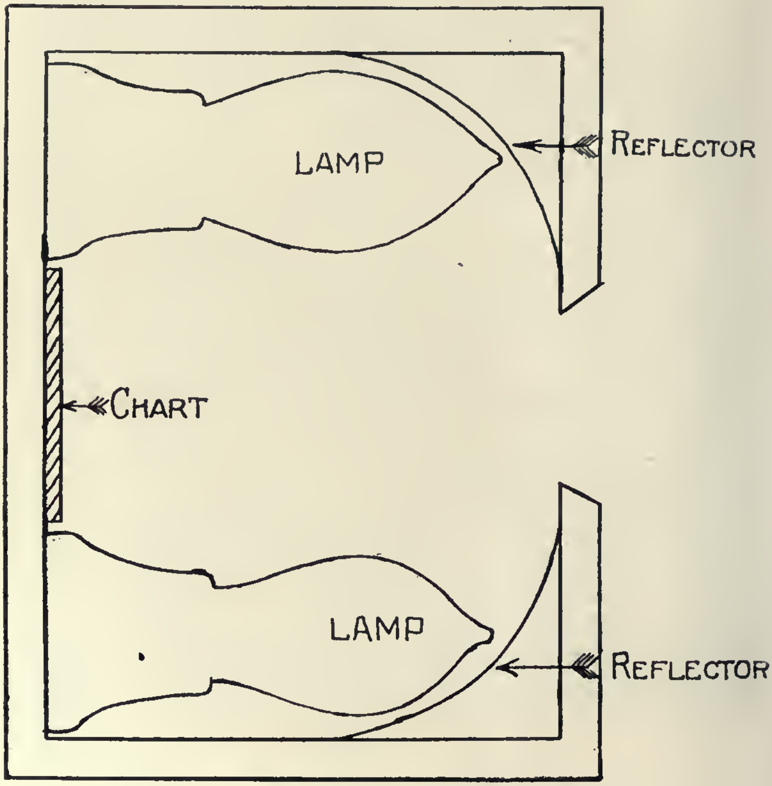

Fig 12.—Rogers' method of illuminating test-type.

and green glass over the examined eye it is readily found if the answers are correct. The circles are for a similar purpose. The electric light below is for muscle test also."

I had the pleasure of seeing this cabinet, while in Grand Rapids, and although it is an admirable chart, to my mind there are two objections to it: First, so many different characters seen at a time are confusing; second, the irradiation from the white illuminated areas is tiring.

The switchboard and rheostat I use with my cabinet also control the illumination of a chart lighted with reflected light from an O'Brien lamp. The two charts are side by side, so I have been able to make direct comparison with all my patients as to the ease and comfort of the two methods of illumination, and the distinctness with which the characters are outlined; without exception the verdict has been in favor of transmitted illumination with black characters on a white ground.

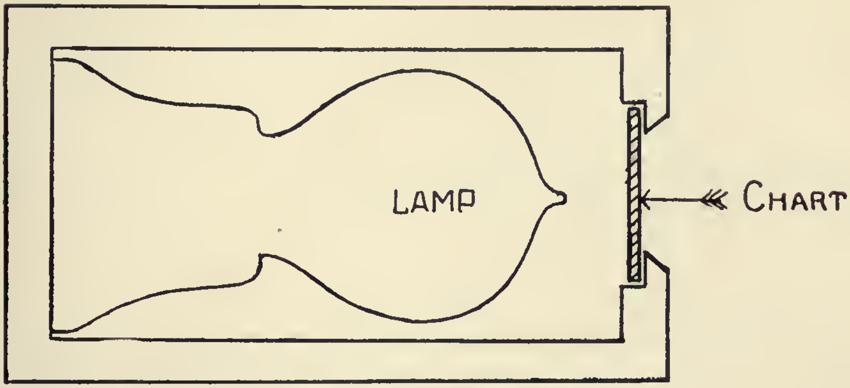

Fig. 13.—Black's method of illuminating test-type.

I wish to express my thanks to Mr. C. R. Gilman, electrical engineer, C., M. & St. P. R. R., for his advice and help in construction of the cabinet, and to the Cutler Hammer Company of Milwaukee for the assistance in building a rheostat and for the loan of the one used in demonstrating this paper.

ADDENDA.

In corresponding with Dr. Rogers, I find the types in his cabinet are illuminated by reflected light as shown by cross-section drawing.

In my cabinet the letters are engraven upon porcelain and are illuminated from behind, as shown by the cut.

DISCUSSION.

Dr. Alt:—Dr. Black has worked out the problem in a more scientific way, but I have been using a lantern in my office for twenty years, which I have never published. I have a lantern frame made of wood, with three illuminating-gas flames in it, and the test letters are painted black on ground glass. I have used this on dark days for twenty years. The only objection to it is that the flame shows through to a certain extent, so that some points are better illuminated than others, and this I have been unable to overcome. After I had used it for several years a similar lantern was described by Dr. Williams of Boston.

Dr. Ryan, Galesburg, Ill.:—If Dr. Black's reasoning is correct, is it not true, then, that the ordinary way of fixing up a board in school is trying to the eyes of the children, by having white letters on a black ground? My impression is that it is almost impossible to see the white on the blackboard at certain angles. Fifteen years ago I recommended that the boards be gray and that black crayons be used instead of white. I have noticed in testing that when the card was old and yellow the patients were more apt to read the letters than when the card is too intensely white. I think if the letters were put on a slightly yellow ground it would much enhance their value.

Dr. Black (closing discussion):—Regarding the flame showing through, evidently the illumination is not properly diffused, and if you will put a piece of ground glass in front of the flame, or a piece of bond paper, it would tend to diffuse the light. The intensity of the illumination would be a little lower, but it would be better diffused. These lights can be obtained from the Ryan Electric Company of Philadelphia.

A FURTHER STUDY OF THE SO-CALLED HOROPTER, MAKING OCULAR ROTATIONS EASY OF UNDERSTANDING.

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(Illustrated.)

In his study of optics, Helmholtz made three fundamental errors from which he was never able to free himself. One of these errors was the construction of the optic axis by beginning at the center of the cornea, making this point the anterior pole, carrying it back through the center of rotation and on to the retina, at a point between the macula and disc, making this point the posterior pole. The second error, growing out of the first, was his faulty construction of the visual axis by carrying it from the macula forward, on the temporal side of the center of rotation, to the nodal point, thence through the cornea to the nasal side of the anterior pole, on into space. The third error, growing out of the first and second, was his conclusion that all lines of direction are the axial rays of cones of light which, he taught, cross each other at the nodal point, a few millimeters in advance of the center of rotation. To these fundamental errors is chargeable the fact that no man, not even his brightest pupil, has been able to understand Helmholtz's chapter on the "Rotations of the Eyes." Even he himself must have recognized the murkiness of this chapter, for, when one of his brightest students said to him, "Professor, I have no trouble with any part of your 'Physiologic Optics' except the chapter on 'Rotations of the Eyes,'" he replied, "I am not astonished to hear you so speak. Leave that chapter alone, for, though it cost me more time and thought than any other part, I must rewrite it; then I hope to make it clear." He lived twenty or more years after this conversation, but never rewrote his chapter on "Rotations of the Eyes." If he had been fortunate enough to have discovered the fundamental errors pointed out above, he would have rewritten this chapter and would have made it so strong and clear that no student could fail to understand. With these errors eliminated, ocular rotations are as easy of comprehension as the simplest problem in geometry, which, indeed, it is.

Helmholtz's greatness made his mistakes more fatal, for the world stood ready to accept his teachings as scientific truths, nor was the world often disappointed. To controvert him, while he was alive, would have been considered audacious, and no one knows better than I do the hard task one has who dares, even now, to teach for scientific doctrine anything contrary to the dictum of Helmholtz. In admiration of the greatness of Helmholtz allow me to say I am second to no man. His portrait hangs on one of my walls and is an inspiration to me. I ought to love him, if for no other reason, because of the three errors from which he could not free himself, otherwise *Ophthalmic Myology* and *Ophthalmic Neuromyology* would never have been written.

In order that the horopter (better the Maddox isogonal circle) may be understood and its full significance may be appreciated, the conditions underlying binocular single vision must be known, for without the power of binocular single vision (impossible to some) there can be no monoscopteric circle.

The supreme law of binocular single vision is the law of corresponding retinal points. The law of direction does not make corresponding retinal points, else the two images of any one point would always result in single vision, regardless of the relationship of the visual axes and the horizontal retinal meridians to the plane of the primary isogonal circle. To illustrate: It must be conceded that a point and its two images lie in the plane of the same isogonal circle, under normal conditions of refraction. A line of direction goes from the image in each eye and the two intersect at the point, and of necessity they both lie in the plane of the circle containing the three points. Each of these lines is a radius of retinal curvature prolonged and, therefore, the point at which they intersect should be always single, regardless of the relative position of the two eyes, if the law of direction creates binocular single vision. If one eye is turned up or down, the point and its two images still lie in the same plane, and the two lines connecting the two images with the one point are radii of retinal curvature prolonged, but the point is not seen single; therefore, there must be a law for corresponding retinal points other than the law of direction.

This law of corresponding retinal points is founded on an anatomic and not on a psychologic basis. If the cones of the central points of the two maculas have not a common brain connection, binocular single vision is impossible. To simplify: Let us suppose that there is but one cone constituting the *fovea centralis* of each macula; the axone going from this cone in the one eye will pass backward toward the brain and will meet the axone from the foveal cone of the other eye, at the beginning of the one tract or the other. One of the axones having crossed, by way of the chiasm, to meet its fellow from the other fovea, they then go side by side to the corresponding cuneus and terminate in one cell in the cortex of that cuneus. The impression of light on these two cones would be a double impression, but these would be transmitted to the one cell and could excite but a single sensation. If the cones of the two foveas have the proper brain connection, it can be easily conceded that there is no faulty connection of the cones of the two maculas, and that the rods and cones of the two retinas, point for point, have common brain connections. The vertical and horizontal retinal meridians cross each other in their respective foveas. If these points of crossing correspond, that is, have a proper brain connection, then the two vertical meridians will correspond point for point, as will also the two horizontal meridians throughout their entire extent. What is true of these two cardinal meridians must be true of any two oblique meridians which bear a similar relationship to the cardinal meridians, as the two meridians at 45°.

If Helmholtz's law of direction, namely, that the lines of direction are axial rays, were true, corresponding retinal points in any two corresponding meridians would have to be measured in millimeters from the foveas, and not in degrees of arc, for the angle of any two lines crossing at the nodal point would neither be an inscribed angle nor an angle at the center of the retinal curve; therefore, it is not measured by either the half or the whole of the intervening retinal arc. A glance at Figure 2 will show this. It is true that the angle B-n-A in this faulty figure, being an inscribed angle, is measured by half the arc B-A, and the angle c-n-b is equal to the angle B-n-A, for they are opposite angles, but it is not true that half the arc B-A is equal to the whole retinal arc c-b. If we must measure the distance of retinal points from the fovea by millimeters, we should measure the distance between spacial points by meters and not by degrees. Most authors who have not rejected Helmholtz's axial-ray theory have been consistent in that they have measured corresponding retinal points in millimeters from the fovea, but have been inconsistent in that they have measured the distance between spacial points in degrees.

Helmholtz was wrong in teaching that axial rays are lines of direction, and this error grew out of his first and greater error, the incorrect location of the poles of the eye. These errors are alike in that he began at the cornea and worked backward to the retina. He should have started at the retina and then worked forward. It must be universally conceded that the retinal meridians all cross each other at the fovea, nor can it be denied that this point of crossing must be the posterior pole of the eye. How easy now to construct the optic axis by extending a line from the posterior pole through the center of retinal curvature (the center of rotation) to the cornea, and thus find the anterior pole, a course directly opposite the one pursued by Helmholtz. The anterior pole thus located is usually to the nasal side of the center of the corneal curve, but in ideal eyes it coincides with the center of the corneal curve. In all eyes the posterior pole is the fovea centralis, but the anterior pole may or may not be the center of the corneal curve. (The real anterior pole is in the aqueous chamber, just as far in front of the center of rotation as the fovea is behind it.) In every eye the antero-posterior axis, or optic axis, prolonged is the visual axis, as distinguished from all other visual lines. This line is a radius of retinal curvature prolonged, and so are all other lines of vision. Otherwise an isogonal spacial circle with corresponding retinal arcs could not be constructed. The true law of direction, therefore, must be this: *"Every line of direction is a radius of retinal curvature prolonged."*

Thus stand corrected Helmholtz's three fundamental errors—first, his error in locating the poles; second, his error in constructing the visual axis, and, third, his error of conception that lines of vision are axial rays of light. One holding to these errors can have no clear conception of the primary isogonal circle (horopter), without which there can be no correct understanding of either monocular or binocular rotations. The chapter that Helmholtz would have written on "Rotations of the Eyes," if these three errors had not made his mind murky, can only be conjectured, but I am willing to concede that he would have surpassed any author preceding or following him.

I was not hunting for Helmholtz's errors when I found the true principles of the horopter or isogonal circle, but the light of the truth revealed his errors. Only a few weeks before his death he dictated a letter to me in which he expressed an interest in my then earlier writings. If he were living now I believe he would count himself happy in that an admirer had pointed out his three fundamental errors, and I know that he would have counted himself thrice happy if he could have found them himself.

Soon after *"New Truths in Ophthalmology"* came from the press I was asked, "Have you read LeConte on *'Sight'?"* I was forced to answer I had not. I procured a copy and read it with eagerness. When I had read "The rods and cones see ends on" and "For every retinal curve there is a corresponding spacial curve," it seemed to me that LeConte not only agreed with me that every line of direction is a radius of retinal curvature prolonged, but that he had antedated me in its publication. I did not feel myself confirmed in this view when I saw his horopter, here reproduced, in Figure 1, from his first edition. I reasoned that the rods and cones all pointed to the center of retinal curvature, and that, "seeing ends on," the line of sight must be a radius of retinal curvature prolonged. I knew that "retinal and spacial curves" could not correspond unless they were concentric, so that a line connecting any two points corresponding in degrees must pass through the common center, hence must be a radius of retinal curvature prolonged. In spite of his faulty horopter (Fig. 1), I was ready to give LeConte credit for a great discovery and so wrote him, sending what I had written on the same subject. To my surprise, his reply stated that he had only spoken poetically and graphically when he stated that "the rods and cones see ends on," for he believed with Helmholtz that all lines of direction are axial rays, and that they cross each other at the nodal point. He further stated that my letter had called his attention to the fact that the horopter in his first edition of *"Sight"* was incorrect in that the circle passed through the centers of rotation (Fig. 1) when it ought to have passed through the nodal points. To be consistent, he had to remodel his horopter, which he did in his second edition of *"Sight,"* and this remodeled horopter is here reproduced in Figure 2, which, as already shown, is not a mathematical figure and, therefore, can not be the true horopter (isogonal circle).

To put it mildly LeConte's disclaimer staggered me and made me doubt for a moment the correctness of what I had published concerning the law of direction. I have no explanation for his holding to Helmholtz's errors after his attention had been called to them and after he apparently had discarded them in the two quotations made above. But further study convinced me more fully that Helmholtz had perpetrated these three fundamental errors, and that LeConte had perpetuated them.

It is cruel to kill a pet with a point. The fatal point I present against the Muller horopter, the Figure 2 from LeConte, is that, if a rotation from A to B is attempted, one of two things must happen: Either the two nodal points will leave the circle, for they are in advance of the center of rotation; or the circle, moving to the left with the eyes, will leave both the first and second points of view. Take either horn of the dilemma, and the value of the circle is destroyed.

Because binocular single vision and both monocular and binocular rotations could not be clearly understood, in the light of the teaching of Helmholtz and a host of his followers, I sought the more earnestly for the correct principles underlying them. These fundamental truths are: (1) The visual axis is the true antero-posterior or optic axis, the posterior pole being the central point of the macula; (2) all lines of vision intersect the visual axis at the center of retinal curvature, which is the center of rotation, and, like the visual axis, are radii of retinal curvature prolonged; (3) the cone at the posterior pole of one eye corresponds with the cone at the posterior pole of the other eye, for the reason that these have a common brain-cell connection in one cuneus; that all other corresponding retinal points are either rods or cones with a common brain-cell connection, either in the one or the other cuneus; that these corresponding points compose the corresponding retinal meridians, and that corresponding points are related, each to its own fovea, by degrees of arc and not by millimeters.

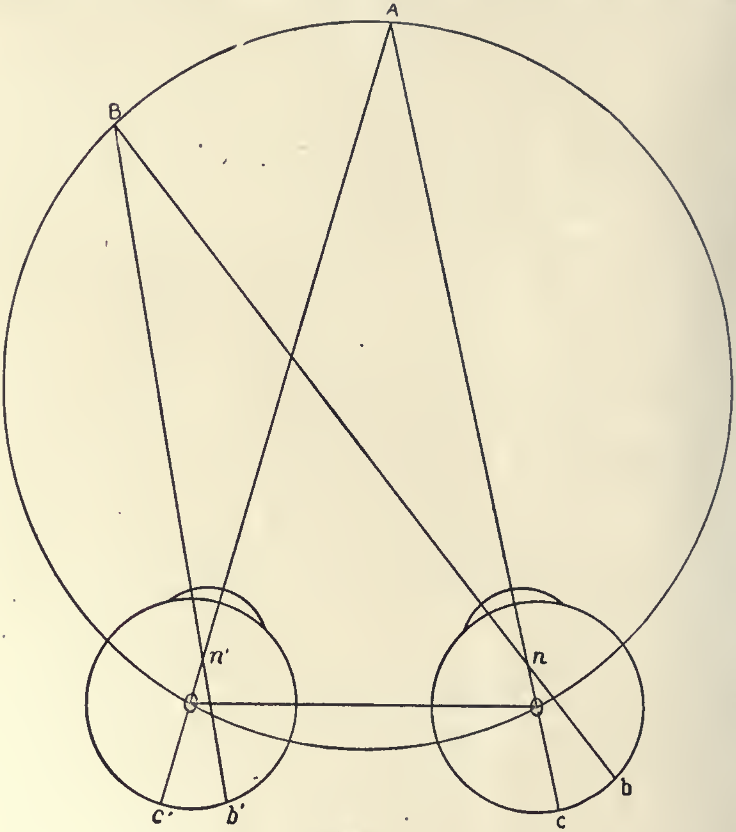

Figure 1.

The above fundamental truths are essential to a clear understanding of binocular single vision and of monocular and binocular rotations.

On the spherical concavity of the retina is founded the mental law of direction, which is : *"Every line of direction is a radius of retinal curvature prolonged."* The spherical concavity of the two retinas and the law of direction create the mathematical circles of possible binocular single vision. These circles are innumerable and must be divided into two classes. Belonging to the first class there is only one circle, the primary, but to the other class belong an infinite number of circles, the secondary. Each of the secondary circles has the same radius as that of the primary circle, and all of these are bisected by the extended median plane of the head. All of these circles have two points in common; that is, the centers

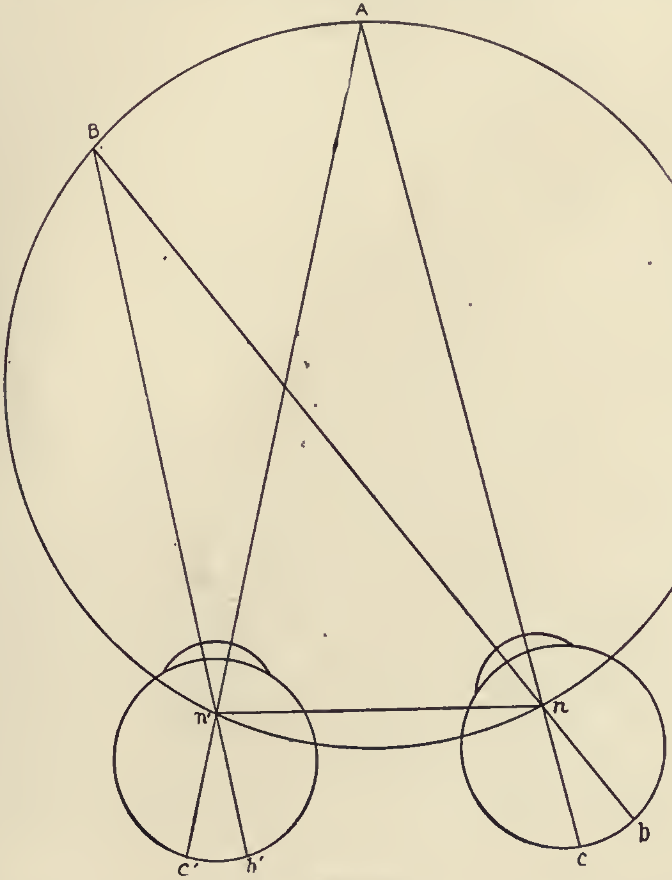

Figure 2.

of retinal curvature which are the centers of rotation of the eyes, but the third point through which each independent circle is constructed lies in the extended median plane of the head, each being the same distance from the center of the cord common to all (the line connecting the centers of the two eyes), as is the point of direct fixation on the primary circle. Each of these circles has its own plane, and all these planes intersect in the cord connecting the two centers of retinal curvature. In each of these planes lie innumerable lines of direction from each eye, one from one eye intersecting one from the other eye at every point on the circle. The angle of intersection for any two lines is precisely the same as the angle of intersection of any other two lines, for each is an inscribed angle and is measured by half of the same arc.

A point on any one of these circles is seen single with the two eyes only when the intersecting visual lines come from rods or cones in the two retinas which have a common connection with a single brain cell in one cuneus. These rods and cones constitute corresponding retinal points. Corresponding retinal points and the relationship of the vertical axes of the eyes to the median plane of the head do not create the isogonal circle. These become monoscopteric circles only when the two visual axes and the horizontal retinal meridians are made to lie in the plane of the primary circle with the visual axes converging at some point on this circle. With one visual axis in the plane of the primary circle and the other in the plane of a secondary circle, either below or above the primary plane, means diplopia everywhere. With the visual axes both lying in the plane of the primary circle, but intersecting within or beyond the circle, means diplopia everywhere. Let the planes of the horizontal retinal meridians fail to lie in the extended plane of the primary isogonal circle, the vertical axes of the eyes either diverging or converging above, can only mean diplopia everywhere,[1] but if the vertical axes should lean towards the right or towards the left, the same number of degrees, there would be no diplopia, but there would be loss of correct orientation.

The law of corresponding retinal points is supreme and imperious, as related to the isogonal circles, both the primary and secondary. The behests of this law are obeyed by the extrinsic ocular muscles, in the interest of binocular single vision and correct orientation. If the two visual axes are made to lie in the plane of the primary isogonal circle, when in its primary position, by watchfulness of the basal centers in control of the superior and inferior recti muscles, and are made to intersect at a point on this circle, at the intersection of the extended median plane of the head, by the action of the third conjugate center on the interni, aided, if necessary, by the basal centers connected with either the externi or interni, and if the horizontal retinal meridians are forced by the obliques to lie in the plane of the primary isogonal circle extended backward, then this isogonal circle is converted into a monoscopteric circle. As a result of this planing and converging of the visual axes, and the planing of the horizontal retinal meridians, every point on all the secondary isogonal circle is seen single; hence these circles all become monoscopteric. The sum of all of these points makes the monoscopteric surface, shown in this model.[2]

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[1] In non-symmetric oblique astigmatism the distortion of images compels convergence or divergence of the vertical axes, in the interest of binocular single vision, but at the expense of correct orientation.

Every point in the field of binocular vision, not in the plane of the primary isogonal circle, lies in the plane of some secondary isogonal circle and also in the extended plane of some retinal meridian, either the vertical or an oblique meridian, and on the line of intersection of these two planes, which is the line of vision for that point. Any one of these points may become the secondary point of view, which means that the two eyes can be so rotated as to bring the two maculas under the two images of the secondary point. In doing this, the visual axes have simply taken the places of the two indirect visual lines which had connected the one point with its two retinal images.

The purposes to be accomplished by the twelve ocular muscles are: first, binocular single vision, with which all of them are concerned; second, range of vision, with which the lateral recti alone are concerned; third, extent of vision, with which all the recti and the obliques are concerned, and, fourth, correct orientation, with which the obliques are chiefly concerned. All of these purposes are accomplished in obedience to a law that governs both binocular rest and binocular motion. That this law may be understood there are three things that must be well in mind: first, the primary isogonal circle; second, the visual axes, and, third, the horizontal retinal meridians. To ignore the first is to fail to comprehend the import of the second and third; hence a knowledge of the primary isogonal circle (the old horopter) is supremely important. It must not be forgotten that to change the point of direct view is to create a new primary isogonal circle, and, since there is an infinite number of points in the line of intersection of the extended median and horizontal planes of the head, there can be an infinite number of primary isogonal circles, only one of which can have a real existence at one and the same time. They are all alike in that each is constructed through the centers of rotation (two fixed points) and the point of direct fixation (a variable point), and that they must lie in a common plane. Likewise in the extended plane of each secondary isogonal circle there may be constructed an infinite number of secondary circles, each differing from the other only in the length of its radius. Still another thing must be known; that is, there is no point in the binocular field that does not lie in either the plane of the primary isogonal circle or in the plane of one of the secondary isogonal circles. And still another fact must not be ignored: that is, the planes of all isogonal circles intersect in the line connecting the centers of rotation of the two eyes, which line is a cord common to all the circles. All of these things necessary for a proper understanding of the law of binocular rest and motion are easy of comprehension, but not easier than is the understanding of the law itself, in the light of these facts.

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[2] The surface exhibited in wood was that generated by revolving the primary circle up and down on the cord connecting the centers of the two eyes, as shown in Figure 3.

The following is the law of both binocular rest and motion:

THE OCULAR MUSCLES MUST SO RELATE THE TWO EYES THAT THE TWO VISUAL AXES AND THE TWO HORIZONTAL RETINAL MERIDIANS SHALL ALWAYS LIE IN THE PLANE OF THE PRIMARY ISOGONAL CIRCLE, AND THAT THE TWO VISUAL AXES SHALL INTERSECT AT SOME POINT ON THIS CIRCLE, IN THE INTEREST OF BOTH BINOCULAR SINGLE VISION AND CORRECT ORIENTATION.

That the above law may be obeyed, every rotation plane must be a meridional plane extended, and the equatorial plane must contain the axis of every rotation. A study of Figure 3 will show that, in direct rotations to the right or left, the plane of the primary circle is not moved, but the visual axes are shifted with unvarying angle in this plane from point to point as from C to A. It will also be seen that in passing from the primary point of view (C) to a point directly above (C′) or below, on a secondary circle, the primary plane (B-C-D) is made to take the place of the secondary plane (B-C′-D), but the visual axes have not been shifted in it. While unmoved in the plane of the primary circle throughout the rotation of the latter, each axis has moved in the extended plane of its own vertical meridian from the primary to the cardinal secondary point of view, the angle of convergence unvarying.

Again, Figure 3 shows that, when fixation is to be changed from the primary to an oblique secondary point (from C to E), the plane of the primary circle B-C-D is made to take the place of the plane of the secondary circle B-C′-D, and the visual axes H-C and

G-C are made to assume the positions of the indirect visual lines E-e and E-e′. During the rotation of the primary plane neither the visual axes nor the horizontal retinal meridians have been allowed to leave it, but both have been shifted in it, each visual axis moving in the plane of that retinal meridian extended in which were located the point and its image before the rotation began.

H, the image of C, is on the macula, at the point of intersection of the vertical and horizontal retinal meridians, while C is on the line of intersection of the extended planes of these meridians. But since the plane of the primary isogonal circle and that of the horizontal retinal meridian coincide, it is easily shown that every object in the binocular field of vision and its retinal image are on the line of intersection of some one isogonal plane and the plane of some one retinal meridian; therefore, the object in the spacial concave occupies the same relationship, in degrees, to the vertical and horizontal retinal meridians extended, as its retinal image in the retinal concave does to these meridians. The line connecting the two is the line of intersection of the extended plane of the retinal meridian on which the image lies and the plane of the isogonal circle on which the spacial point lies. Since the plane of every isogonal

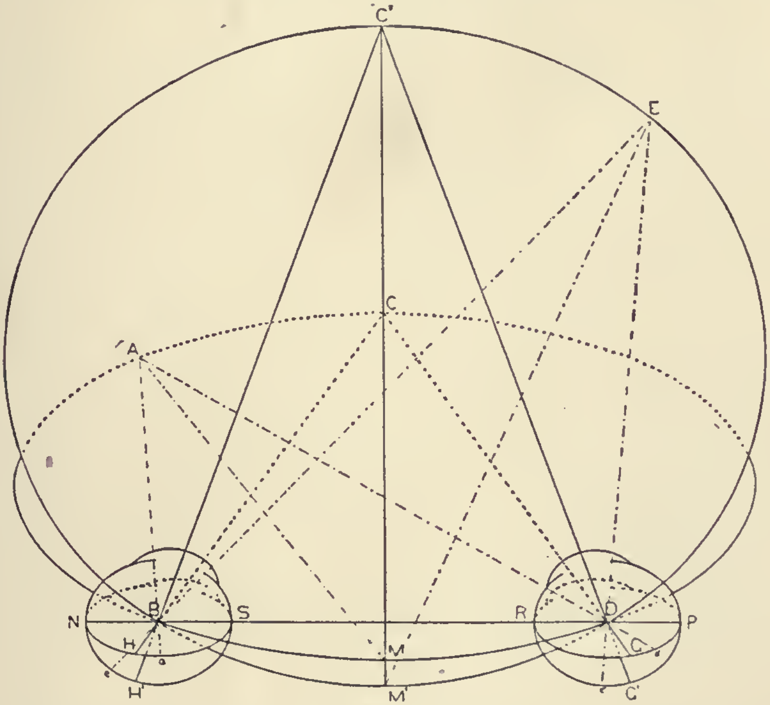

Figure 3.

circle as well as the plane of every retinal meridian passes through the center of rotation, their lines of intersection, which are lines of direction, must all pass through the center of rotation. This is another proof that every line of direction is a radius of retinal curvature prolonged.

It only remains now for me to show that every member of my large family of cortical and basal brain centers is real and not imaginary. This I can do by a further study of Figure 3. In this figure, C is the direct point of fixation on the circle B-C-D. In both intrinsic and pseudo-esophoria, the visual axes would tend to cross at some point within the circle, but this too early crossing is prevented by neuricity sent from some center or centers in the brain to the two externi. Since there is no divergence voluntary center in control of the two externi, each of these muscles must be controlled by an individual involuntary center at the base of the brain, these are the right and left fourth basal centers, duction centers for the externi and under control of the fusion faculty of the mind. These two centers send just enough neuricity to their respective externi to compel intersection of the visual axes at C.

Again, if there is exophoria, the visual axes would tend to cross at some point beyond C, but the intersection beyond the chosen point of view is prevented by the two basal fusion centers for the two interni, these fusion centers, the right and left third basal centers, supplementing the neuricity from the third conjugate or convergence center so as to force intersection of the visual axes at C.

If the point of fixation of the visual axes of esophoric eyes is to be changed from C to A, on the same circle, three brain centers, two of them conjugate and one basal, must participate. Convergence at the same angle must be maintained by the third conjugate center, and the visual axes must be moved to the left by the left externus and the right internus, these being stimulated to do so, in part, by neuricity from the fifth conjugate center, but in part also by neuricity from the left fourth basal center to the left externus. One of the three muscles concerned, namely, the left internus, in this changing of the points of view, is under a single stimulus, the source being the third conjugate center. The right internus receives an equal convergence stimulus, but it also receives neuricity from the fifth conjugate center, the purpose of which is to rotate the right eye directly to the left. The left externus also receives neuricity from a double source, the one source being the fifth conjugate center, the other source being the left fourth basal center. Except for the supplemental neuricity from the left fourth basal center, the left visual axis would not move so fast as the right;

hence they could not reach A at the same time, nor could the same angle of convergence be maintained throughout the rotation. It can not be denied that a weak muscle must have more neuricity than a strong muscle for the accomplishment of a given work. A conjugate center gives equal quantities of neuricity to the two muscles under its control; hence if one of a pair is weaker than the other, there must be an individual fusion center for the weaker muscle, from which must come the supplemental neuricity.

In the next place, granting that there is lateral orthophoria, there is a need for individual basal fusion centers in all cases of hyperphoria. Let there be left hyperphoria and right cataphoria, of each 5°, with C as the point to be fixed. The interni are ready to respond perfectly to neuricity from a single center, the third conjugate. Tonicity of the superior and inferior recti of the left eye would place its visual axis in the plane of a secondary isogonal circle elevated 5° above the plane of the primary circle; the tonicity of the superior and inferior recti of the right eye would place its visual axis in the plane of a secondary isogonal circle 5° below the primary plane. Diplopia must follow without intervention of nerve force, and whence comes this force? Not from the first conjugate center, for this would elevate both eyes. Not from the second conjugate center, for this would depress both eyes. In neither case would the two axes be placed in the primary plane. To prevent diplopia the right first basal center must send neuricity to the weak superior rectus and thus make it elevate the visual axis into the primary plane; and the left second basal center must send neuricity to the weak inferior rectus and thus make it depress its visual axis into the primary plane. Thus the visual axes are brought into the plane of the primary isogonal circle by the two basal fusion centers, allowing the third conjugate center to converge them at C, with binocular single vision as a result.

In every possible binocular rotation the ocular muscles have one common task to perform, namely, the keeping of the two visual axes and the two horizontal retinal meridians in the plane of the primary isogonal circle, and the converging of these axes on some point on this circle, thus making of the single primary isogonal circle and the infinite number of secondary isogonal circles, all monoscopteric circles, or circles of binocular single vision.

The centers concerned in any rotation of any pair of eyes whether these eyes be orthophoric or heterophoric, can be known, and the work done by these centers can be understood by any oculist who sets himself to the task. It would be a pleasure to me to point them out, but this paper has already grown too long.

The aim of the treatment of all heterophorias is to give equal tonicity to the two muscles of every pair so that the basal or fusion centers may have no abnormal work to do in planing the visual axes and horizontal retinal meridians and in shifting and converging the visual axes at a point on the primary isogonal circle.

Listing's plane and law, Helmholtz's poles, law of direction and nodal point, Muller's horopter, and the multitude of errors growing out of these must be forgotten or ignored if one is to have any accurate knowledge of ocular rotations. The same is true if one wishes to know how to detect and deal with heterophoric conditions.

DISCUSSION.

Dr. Reynolds, Louisville, Ky.:—The differences between the optical axis of a lens, its line of illumination, and the line of collimation in a compound system, were fully described and illustrated by Sir John Herschel, whose "Outlines of Astronomy" was one of the text-books of my schoolboy days. The same principle is involved in the determination of zenith and azenith that Dr. Savage employs to elucidate his axial and antipodal lines. The collimator serves all the necessary purposes of demonstration in physiological optics as in practical astronomy.

THE PROPHYLAXIS OF OPHTHALMIA NEONATORUM.

F. Park Lewis, M.D.

BUFFALO, N. Y.

It is, of course, not necessary to discuss before the Academy the scientific aspect of ophthalmia neonatorum. If the subject is once opened as to which is the better of the various methods of prophylaxis or treatment that may be employed, it will at once divert attention from the main point, i. e., that prophylaxis is efficient and that early treatment is almost absolutely sure in its results. If no other measures were available than a sanitary toilet of the eyes of the child and nitrate of silver properly applied, eyes need not be lost. When, therefore, we learn that of the number of children admitted into the asylums and schools for the blind last year one-quarter had lost their sight from this cause, and that at Overbrook, a suburb of Philadelphia, the average for the past sixteen years was 29 per cent. of the whole, it becomes evident that ordinary sanitary precautions are not being employed and that more effective measures must be taken to control so serious a menace to the well-being of our citizens.

The widespread nature of this neglect is shown by the census reports of the special commissions for the blind in New York and Massachusetts. In the former state the total number of blind persons reported was 6,200. Of these, 569 were under 1 year of age and under 4 years, including those under 1 year, were 959 children, while in the state of Massachusetts among 3,306 blind registered 661, or more than 20 per cent., had become blind before their fifth year. If we exclude ulcerative conditions due to bad hygiene and insufficient nourishment, and congenital blindness, which in many instances may be avoided by preventing the congenitally blind from mating, we may safely assume that one-half of this number, or 10 per cent. of the whole, have given their eyes as a tribute to ignorance and neglect. Such figures as these, in which so large an amount of blindness is produced every year as the result of an infection that should never have occurred, emphasize, first, the necessity of a clear understanding of the conditions producing these infections; and, second, the necessity of an organized and concurrent movement for their control.

Said Dr. Joseph Price of Philadelphia a week ago, in discussing this subject at a meeting of the American Obstetrical and Gynecological Association: "The place to begin in the prevention of ophthalmia neonatorum is with the man who gives gonorrhea to his wife, for in 60 per cent. of the cases the gonococcus is found." Every young woman is entitled to know the danger which she incurs both for herself and her future children when her husband brings to her so virulent an infection.

"The important thing," said Dr. Juan Santos Fernandez of Havana, "is to bring before the public mind by means of constant propaganda a knowledge of the danger to a recently born child who is at all affected as to the eyes, the great harm which a husband affected with gonorrhea may cause his wife or offspring, and side by side with these to call the attention of the family to the facilities which the authorities will furnish them to guard against blindness. This," he continued, "would be worth much more than penalties, and if there were a physician paid by the state (and in every county in the United States may such a health officer be found) to attend to the poor children affected or to prevent their becoming affected, they would surely seek his assistance and he could fulfill his duties."

At a great meeting at Boston said Helen Keller a short time since, speaking of blindness due to ophthalmia neonatorum: "The problem of prevention should be dealt with frankly. Physicians should take pains to disseminate knowledge needful for a clear understanding of the causes of blindness. The time for hinting at unpleasant truths is past. Let us insist that the states put into practice every known and approved method of prevention and that physicians and teachers open wide the doors of knowledge for the people to enter in. The facts are not agreeable reading; often they are revolting, but it is better that our sensibilities should be shocked than that we should be ignorant of facts upon which rest sight, hearing, intelligence, morals and the life of the children of men. Let us do our best to rend the thick curtain with which society is hiding its eyes from unpleasant but needful truths."

The second point is to require sanitary decency on the part of those having to do with the parturient woman.

As the distinguished surgeon and citizen of this town, Dr. McMurtry, has said, "Every case of childbirth is a case of surgery."

There is no condition in which a woman can find herself which calls out more helpful sympathy on the part of womankind everywhere than that of pregnancy. There is no condition which the wives and mothers of the poor find more deplorable than that of childbirth. Compelled by motives of economy, they seek in the hour of their trial the aid of a midwife. She is often untrained and unclean. Listen to the description given by Elizabeth Crowell in a recent number of *Charities and the Commons of the Midwives of New York*. Of an Italian midwife whom she visited she writes: "Her home was of the dirtiest, the condition of her hands was indescribable, her clothing filthy, her bag beggered description. As to the midwives' homes, 106 were absolutely filthy, as was the clothing and person of the midwife herself. As for the bags and their equipment, from a professional standpoint, by far the greater number would make fit decorations for a chamber of horrors. Rusty scissors, dirty string, a bit of cotton, a few corrosive sublimate tablets, old rags and papers, some ergot and vaselin, a gum catheter wired, were the usual contents."

There are a thousand of these women in New York and they were present last year at the accouchement of 43,834 mothers, or 42 per cent. of the whole number of births. As a result, says Dr. Jacoby, septic peritonitis is exceedingly common and deaths frequent.

How much blindness results from ophthalmia neonatorum under such ministrations no one can estimate. But when we find in a home for blind children that 12 out of 16 have lost their eyes from suppurative inflammations, the necessity for urgent action is self-evident.

The first essential, then, for the betterment of these conditions is broader enlightenment concerning them. It is not a medical subject only. This is one of those forms of disease, like tuberculosis, which affects the entire community. It is a matter of great social importance, and social workers, when once the subject is understood, will prove to be important factors in effecting its control. But they must know what it is; how it is acquired and how it is avoided. It must be made clear, too, that infections are not always gonorrheal, nor do they always occur at the birth of the child. Even physicians sometimes forget to advise the mother and nurse of the danger of a lochial discharge getting in the child's eyes through the use of the same bath water or sponges that have been used for the mother. It is evident, then, that two things are necessary: First, a regular and systematic campaign of education carried on through properly constituted authority. This should obviously be the work of the department of public health.

Circulars of advice should be issued from time to time, giving mothers and midwifes, in the simplest language, directions as to the necessary precautionary measures that must be taken if dangerous infections of the eyes of the child are to be prevented. This is very generally done by the Valentin Hauy Society in France, and the leaflet issued by this organization is one of the best published. It points out the danger to the new-born child of infections—the necessity of repeated vaginal douches by the mother up to the time the child is born—the manner in which the child's eyes should be cared for as soon as the head is born, and in heavy black letter type is urged the immediate necessity of putting the child under the care of an oculist if the eyes become at all inflamed. Again, physicians are not all well informed as to the best methods of treatment to follow. The silver salts are used inadvisedly. When this subject is brought to the department of public health in each state for effective action the first inquiry will be, What should be done and how should it be done?

Would it not be better for this Academy to speak authoritatively rather than to jeopardize the whole movement by the issuance of matter which we might hesitate to endorse?

It would seem at present impossible to *eliminate* the midwives, desirable as that might be. They are the product of the custom of ages, but while they continue to do their vitally important work where in every instance two human lives and the child's eyes depend upon the cleanliness, in the interest of humanity and decency this must be secured. They should gradually be replaced by trained physicians and there ought not to be any difficulty in this particular in college towns from which so many young men go out without adequate obstetrical training, but meanwhile they should be put under the absolute control of the department of public health.

The second important essential, therefore, would be to put these women under the authority and control of the health boards everywhere until gradually they can be replaced by trained physicians. They should be licensed, registered, kept under strict surveillance and required to register each birth and report each case of ophthalmia neonatorum within twelve hours after it occurs. They should be advised how to manage the toilet of the eyes immediately the head is born. They should be supplied with a safe prophylactic by the state board of health and carefully advised as to how it should be employed. Failure to use it and to report that it has been used when ophthalmia follows with loss of or injury to the sight should subject the offender, when negligence can be shown, to loss of license and fine. Dr. Oscar Elbrecht of St. Louis made the excellent suggestion at the meeting at Detroit that physicians should be delegated by the health officer to visit each woman delivered by a midwife to be assured that she and the child were left in a normal condition.

I fear that this would be considered an invasion of personal liberties and would be resented by the more independent and intelligent of our citizens, but it is certainly worthy of consideration.

The conclusion, then, that would seem to be warranted from the existing conditions are:

First.—Responsibility. That the responsibility for the control of ophthalmia neonatorum rests with the state and should find its expression through the department of public health.

Second.—Duty with the medical profession. The duty of pointing out its dangers, its prevalence, its prophylaxis and its treatment and suggesting measures for its relief through the proper channels remains with the medical profession.

Third.—Propaganda, impersonal but authoritative. To wipe out this disease as a cause of blindness, the public must be better informed concerning it. They can be reached through various social organizations. The material to be used should come from some authoritative body like this Academy.

Fourth.—Legislation. To accomplish effective work, a concerted effort should be made to secure uniform laws governing the midwives in the several states and in federal territory. Laws should be enacted such as already exist in many European countries requiring their examination, licensure and registration. They should be under the direction and surveillance of the department of public health. Should immediately report every birth and every case of ophthalmia neonatorum when it occurs, as is now done under the Howe law in many states, and should state upon the birth certificate whether or not a prophylactic was used. In case this statement does not appear, then the development of ophthalmia with impairment or loss of sight of one or both eyes of the child without satisfactory explanation should be sufficient warrant for the forfeiture of her license and fine.

If the midwife is to be held responsible for a failure to employ prophylaxis, it is only just that a pure and safe preparation be put in her hands. Although neither the special microbicide nor the strength to be used should be specified in any legislative enactment, it would be quite proper for this Academy to speak authoritatively for the guidance of health boards who are sure to seek advice. It should be urged, moreover, that health boards require the routine employment of prophylaxis in all lying-in hospitals, almshouses and other public institutions and advise its use in all suspected cases in private practice.

It should be made clear that excessive reaction follows the use of the 2 per cent. solution of silver nitrate only when too much of the solution has been employed, as Hubbell has pointed out, or when the salt is impure, containing free nitric acid, as Alleman, Hiram Woods and others have shown. This was first pointed out by Dr. Squibb and is of great importance. A single drop should be carried on a small glass rod of a solution made from the fused silver. Silver catarrh will not then follow except in premature infants where the vulnerability is greater, when a weaker solution with extreme care that asepsis be maintained is safer and better.

The centralization of the authority for the control of the midwives in the State Department of Health would necessitate an examining board and registry for these women in every county of each state. On each of these should be a health officer, a physician whose practice is largely obstetrical, and an ophthalmologist. The appointment of these examining boards, if they were constantly stimulated to effective work, would be of great educational value to all practitioners who might otherwise be inclined to become negligent or indifferent, and it is doubtful whether further efforts would be required to keep the members of the medical profession up to a full realization of their duty and responsibility.

REPORTS OF BOARDS OF HEALTH.

For many reasons it might prove burdensome and ineffective to exact from the physician a report of each case of ophthalmia that occurred in his practice, but it would be entirely feasible and the data would be of great value if at stated periods of six months or a year return postcards were sent to all persons engaged in the practice of obstetrics asking for a report on the number of cases of ophthalmia neonatorum that have occurred in their practice during that period, what prophylactic, if any, had been used and with what results in the case of each eye. If upon these cards the value of prophylaxis is emphasized and its employment urged the educational value of this procedure at infinitely small cost would be beyond estimate. It would be eminently desirable, too, if the department of public health were to insist upon accurate records being kept in all public institutions in which lying-in cases are accepted, as to the number of cases of ophthalmia neonatorum that had occurred, the prophylaxis used and the results. In every one of these cases some prophylactic measure should be required.

ORGANIZATION.

A disease occurring sporadically and which is endemic can be controlled only by organized and concerted effort. It is most important, therefore, in order that no false move be made, that the procedures to be determined upon should originate with the ophthalmologists and obstetricians. Their practicability must be assured by those expert sanitarians engaged in public health work. Then the measures recommended should be carried out by an organized movement in every state in the Union. A state committee should be appointed in each state and through that a like committee in each county. This latter body would ultimately become the board of examiners for midwives. It would be most desirable if in every instance the local health officer should be a member of this committee. Such a general and concerted effort made throughout the country would in a comparatively short time so limit infections and improve methods of treatment that the disasters following ophthalmia neonatorum would practically cease, that this prolific cause of blindness would be controlled, millions would be saved to the commonwealth and the happiness and efficiency of humanity enormously augmented.

REPORT OF THE COMMITTEE OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY APPOINTED TO COOPERATE WITH THE COMMITTEE OF THE AMERICAN MEDICAL ASSOCIATION ON LEGISLATIVE MEASURES TO PREVENT OPHTHALMIA NEONATORUM.*

DR. BAKER:—I would preface the report by a few words to state that the whole country as well as the profession is greatly indebted to Dr. L. Howe for the excellent work he has done in educating us to this work. In Ohio, a number of years ago, under his stimulation, a committee was appointed to go before the state legislature to secure a law for the prevention of blindness. The law provided that every midwife meeting a case of ophthalmia neonatorum was obliged to report same to a physician. The penalty for failure to make such a report is a fine of $50 and six months' imprisonment. It did not provide for the placing of the enforcing of this law under the board of health, and that is the weakness of the Howe law. During the first year I went to the health office and had a circular issued and distributed to physicians and midwives. We also published articles in our medical journals and in a few newspapers. I think for a few months it may have been indirectly of benefit, but no one was ever arrested for its violation. At present I doubt if any midwife in the state knows there is such a law on the statute books. For that reason the committee thought it best, after due consideration, to place this in the hands of recognized authorities to enforce these laws rather than let it go in a haphazard way and leave it in the hands of professional men to see that it is enforced.

Mr. President:—Your committee presents the following for the consideration of this Academy:

It is well known that ophthalmia neonatorum is the cause of a large proportion of blindness in the United States. No fact in medicine, however, has been more thoroughly established than that it is a preventable disease in a vast majority of cases. Granting this, your committee submits that every state should enact laws having for their purpose the adoption of measures for the prevention of its development, at least, in public institutions where obstetric cases are received, and in the practice of midwives. Precedence has long existed in enactments requiring the carrying out of certain methods and treatment to prevent smallpox and other infectious diseases.

The state department of health seems to be the proper authority for establishing the necessary rules, regulations and ordinances and enforcing their observance, and for publishing and distributing appropriate information and instruction on the subject.

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* After the reading of Dr. Lewis' paper the report of the committee appointed last year was requested, and Dr. Hubbell, its chairman, being absent, Dr. A. R. Baker, of Cleveland, read the report.

This committee, as will be seen by the draft of law hereby suggested, is of the opinion that legislative action on this subject should, at present, be limited to the obstetric practice in public or semi-public institutions and to the practice of duly licensed midwives, otherwise leaving the use of prophylactic measures to the judgment and discretion of physicians.

This committee further believes that the specific methods to be followed in the prophylaxis of ophthalmia neonatorum should not be stated in any law, but should be left to the intelligence and judgment of the department of health of each state respectively, presuming that it will act in accordance with the most advanced existing knowledge.

The following form of law is recommended to the committee of the American Medical Association as a basis for legislative action to be modified to suit the needs and legal situations in each state, and the members of this Academy are urged to cooperate, both individually and collectively, with said committee to secure its enactment:

An Act to Prevent Inflammation of the Eyes of the New-born Babe, or So-called "Ophthalmia Neonatorum."

Section 1.—The department of health of this state is hereby vested with power and authority to publish and distribute such information and instruction, to furnish such remedies, and to make such rules, regulations and ordinances as it may deem expedient to prevent the development of inflammation of the eyes of the new-born babe, or so-called ophthalmia neonatorum, in public hospitals or institutions in which midwifery is practiced either wholly or in part, and in connection with the practice of legally licensed midwives.

Section 2.—Said department of health is authorized to enforce its rules, regulations and ordinances at the expense of the state.

Section 3.—Any person violating any rule, regulation or ordinance of said department of health regarding the prevention of ophthalmia neonatorum shall be guilty of a misdemeanor.

This act shall take effect immediately.*

Alvin A. Hubbell, Chairman.
Albert R. Baker,
Edward Jackson,
Committee.

DISCUSSION.

Dr. Alt, St. Louis, Mo.:—Dr. Lewis, in mentioning the use of nitrate of silver in a prophylactic way, did not explicitly state that Credé's method applied correctly consists in one drop of the solution being dropped from a glass rod on the cornea. Credé stipulates absolutely on the center of the cornea, for the reason that from there it will gradually go in all directions and reach all parts of the conjunctival sac. In teaching physicians this method, and in comparing results, Credé's own teachings should be closely adhered to.

Dr. Green, Jr., St. Louis:—In Missouri we have a law and have experienced the same difficulty that Dr. Baker alludes to in Ohio. The law provides that midwives should report to legally qualified practitioners within a certain period. As we know, midwives are frequently in collusion with doctors, and it is not difficult to say, "I reported that case to Dr. So-and-So." The report should be made, and it was suggested by Dr. Shirk, to the local boards of health.

Dr. Moore, Huntington, W. Va.:—With regard to using nitrate of silver, I find that general practitioners are sometimes careless about their solution. Some time ago I was called in consultation and the physician said, "I used the nitrate of silver and it did not do any good." I tore the paper off the bottle and there was a black precipitate and I tested it and found no silver. I have found a I per cent. silver solution efficient, and you can keep it by adding a drop of dilute nitric acid. It makes it more irritating, but no more so than a 2 per cent. solution would be. I have tested this in gonorrheal infections in the adult with good results. I would like to say a word about argyrol. Men who have been using it in 50 per cent. solution have been having good results, but those using a 25 or 30 per cent. solution have not had results. We must remember that while weaker solutions kill the germs in the laboratory, when dropped into the eye it is rapidly diluted, hence a 50 per cent. solution must be used to accomplish anything.

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*The Academy adopted this report as a whole and commended the committee for its work. The committee is to report to the A. M. A., which has taken up the question of ophthalmia neonatorum legislation.

DR. JACKSON, Denver, Colo.:—When we begin to talk of methods of prophylaxis we lose sight of the point of the paper, and what the committee desires to bring before the Academy. That is the matter of getting public supervision, of getting effective machinery for applying what we know about prophylaxis. The point here is the placing of ophthalmia neonatorum, with other contagious diseases, under the care of the boards of health; charging them to look after the good of the community in this respect, just as with smallpox. It seems to me a distinct advance in the matter of legislation. The form of law proposed might not be adopted by any state; it may only take the shape of the addition of a phrase to the general laws governing boards of health; and yet that would probably be more effective than any special act directed to this particular subject. Special acts, as Dr. Baker and Dr. Green have mentioned, after a time will be forgotten and fail to be at all effective. I hope the discussion will stick to the point of the paper.

DR. BROWN, Columbus, Ohio:—As oculist to the Ohio Institution for the Blind, I am convinced that the committee has the right point of view in its work, which is to awaken the profession to the tremendous importance of this subject and to this disease as a cause of blindness, and through this to have the profession educate the people at large to a recognition of the same. The Ohio law was passed in March, 1894, and provides a penalty for failure to report these cases. In two months after the passage of the law I had under observation one of the sad cases of loss of sight from neglect to report the disease or to have a physician take charge of it until complete ulceration of the cornea had occurred. I reported that case to the health officer of the city, one of our most intelligent practitioners, and asked him to bring prosecution under the act. He said, "It will simply start trouble among those Germans in the south end and do more harm than good. Let's put a piece in the paper and call attention to it." And that is the kind of handling this law has had. Many of the profession of the state are not aware of the existence of the law. The people are not to blame, but the profession. The local physician who puts in nitrate of silver and leaves the case for three days is neglecting it. In 1894 we had a falling off in the number of cases entering our institution and I was in hopes the effect of the law was going to be shown in our statistics, but it has again come up to the usual standard; and, in 510 cases examined in the past ten years presenting themselves at the Institution for the Blind, 90 have been due to ophthalmia neonatorum. If I would include 120 previously examined, the percentage of the total would run up to 23. Now, we must remember that these cases represent only those malignant types of the disease, and for every one case that came into the Institution for the Blind there must be many cases of defective vision due to less virulent lesions of the disease, but who go through life handicapped because of the same. And is this not due to a failure of the medical profession to appreciate its moral responsibility in this matter? I hope for the continued agitation of this subject, not only until we get laws that will insure a proper protection to the new-born from the lesions of this disease, but laws that will be so appreciated by every member of the profession that no means will be neglected looking to their enforcement, and consequent prevention of blindness from ophthalmia neonatorum.

Dr. Faith, Chicago, Ill.:—I think we will accomplish much by teaching our students. Several years ago I impressed on my students that one of the questions in examination would be on this subject and that the man who did not answer that would not pass. It is one of the important things they must know. If nothing else were taught but the recognition of ophthalmia neonatorum by the student much good would be accomplished.

Dr. Young, Burlington, Ia.:—I agree with Dr. Brown on this question, that while the law is all right the trouble is in enforcing it. I have never known the law to be enforced in Iowa, although it has been on the books for twenty years. The real fault lies with the profession. I have the misfortune to be a teacher in a school for nurses, and I teach these nurses that they must do certain things, but when the teacher in obstetrics comes in he tells them "there is no necessity to do as Dr. Young says; it is sufficient to wash the eyes out and have them clean." I tell them that in the different classes of people they are serving they are liable to find ophthalmia neonatorum in one as well as another, and they should take no chances. Credé's prophylaxis, and I insist on his exact technic, has abundantly shown its worth.

Dr. Lewis (closing discussion):—I made no reference purposely to the kind of prophylaxis, although it seems to me the Academy, through this committee, might aid the health boards by a statement on this subject. In almost all cases reported in Buffalo protargol had been used as a method of prophylaxis. Nevertheless, infections occurred. The necessity of an efficient prophylaxis must be made clear. The health boards will ask us what kinds of circulars should be used; what should be said to the midwives. If the health officers are going to direct their batteries on the midwives they will want some authority which is unquestionably accepted. I would suggest that this committee of the Academy might take as a basis the Valentine Haüy circular, making any necessary modifications so that there will be some authority to which they may go, knowing that they are doing the right thing. As Dr. Brown remarked, I think we cannot emphasize too strongly the necessity of putting into the hands of midwives the things we want them to use. Physicians have sometimes failed to bring nitrate of silver in their bags, and in these cases ophthalmia neonatorum has developed. The health boards should know what to do. We should like to have authority given the committee so there will be, when the proper time comes, those who will carry on this work and will know both what to do and how to do it. The obstetricians have already taken this up. At the meeting in Detroit ten days ago the matter was thoroughly considered and a committee appointed to do just the work that the ophthalmologists are doing. Next week the same work will be taken up by the Public Health Association in Atlantic City. If, then, the ophthalmologists and obstetricians will determine upon methods, the health authorities will be prepared to put their recommendations into effect; and, aided by the organization of the American Medical Association, a plan, when devised, may be carried into effect throughout the country.

DEFECT OF ABDUCTION ASSOCIATED WITH RETRACTION OF THE GLOBE IN ADDUCTION.

JOHN GREEN, JR., M.D.

ST. LOUIS, MO.

(Illustrated.)

Various authors have described a congenital anomaly of the ocular muscles of which the most conspicuous signs are: (1) Partial or complete defect of abduction; (2) some limitation of adduction, and (3) retraction of the globe into the orbital cavity. The condition does not appear to have been generally recognized as a clinical entity, most authors resting content with a simple description of the anomaly, without venturing into the field of speculation with reference to its causative factors. It remained for Duane[1] to collate all reported cases and, in a masterly analysis, to establish once and for all this symptom-complex as a definite clinical entity. His paper, which is a model of painstaking and careful research, has thrown much light on the causes underlying the condition.

The typical features of the syndrome, as summarized by Duane,[1] are as follows:

"1. Complete, or less often partial, absence of outward movement in the affected eye.

"2. Partial, or rarely complete, deficiency of movement inward of the affected eye.

"3. Retraction of the affected eye into the orbit when it is adducted.

"4. A sharply oblique movement of the affected eye either up and in, or down and in, when it is adducted.

"5. Partial closure of the eyelids (pseudoptosis) of the affected eye when it is adducted.

"6. Paresis, or at least marked deficiency of convergence, the affected eye remaining fixed in the primary position, while the sound eye is converging."

At the time of the publication of his paper (March, 1905) Duane was able to collect 54 cases, partly from the literature and partly through personal communications of ophthalmic colleagues. In view of the fact that Duane himself observed no less than six examples of the anomaly, it seems probable that the condition is less rare than the paucity of cases in the literature would indicate, and that it has been overlooked or incorrectly interpreted by observers less discerning than this distinguished author. My belief is that were the condition thoroughly understood by ophthalmologists generally the number of recorded cases would soon be greatly increased and certain obscure points relative to the precise mode of causation of the unusual movements of the globe would be definitely determined.

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[1] Archives of Ophthalmology, March, 1905.

Two years ago it was my good fortune to encounter a typical example of the anomaly in question, and I venture to present an account of the case, together with photographs, which, better than any description, indicate the special features of the syndrome:

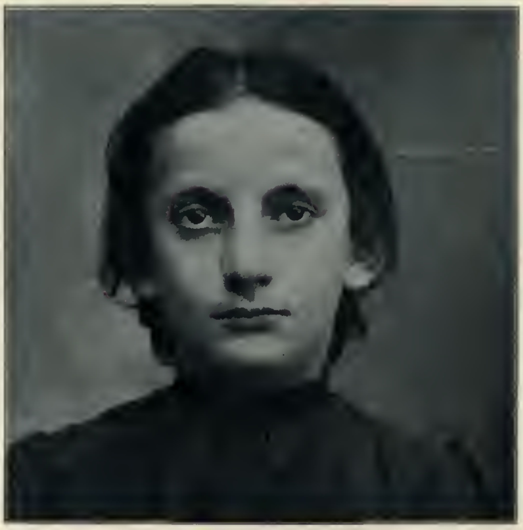

Figure 1.

Dec. 20, 1905, Mollie G., aged 9 years, came to my service at the Jewish Hospital Dispensary. The father, mother, two brothers and three sisters have no ocular peculiarities. One brother, who died at the age of 7 years, is said to have had "cross-eyes."

The mother has always noticed something "queer" about the child's eyes. Her general condition is excellent, and mentally she is on a par with children of like age. She has never suffered from headaches or asthenopic symptoms and is brought to the dispensary simply because the eyes "look queer."

The patient holds the head slightly to the right, with the chin directed toward the left shoulder. With the eyes in the primary position (Fig. 1), the left is enophthalmic 1 mm. and divergent about 5 degrees. Each eye diverges slightly under cover. Fixation is habitually with the right eye. Tests indicate the absence of binocular vision. Diplopia cannot be evoked in any portion of the field of fixation.

R. eye H. 1.5, V. 16/15.
L. eye H. 1.5, V. 16/24.

Pupils react normally to light and convergence. The eyegrounds are normal.

Rhinoscopic examination by Dr. W. M. C. Bryan is as follows: "Uvula enlarged and bifurcated. Right nares occluded. Left middle turbinate enlarged and pressed against the septum, the upper portion of which is deflected to the right. The left lower turbinate is moderately enlarged, but does not encroach on the septum. The right lower turbinate is very much enlarged, fitting the entire space. It can be contracted with adrenalin so that the patient is able to breathe on that side, but the opening thus secured is not sufficient to give a good view of the upper and middle meatus."

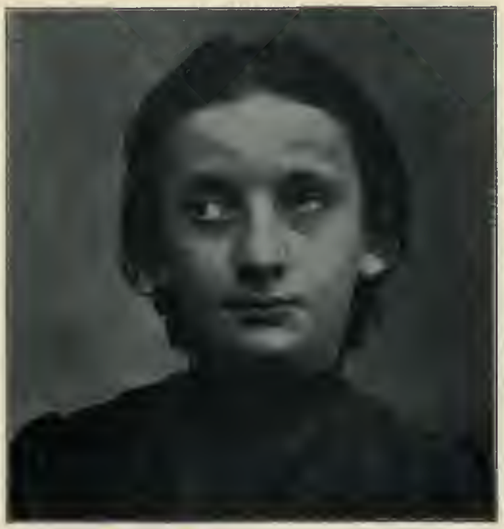

Figure 2.

The difference in the width of the palpebral fissures is well shown (Fig. 1), the right measuring 8 mm., the left 6 mm. When the eyes are directed to the right the patient presents the appearance shown in Figure 2. The right eye makes a full excursion to the right, its palpebral fissure widening to 9 mm. The left eye turns 15 degrees to the right in the horizontal plane and then shoots obliquely up and in. Its excursion terminates with the inner limbus, 3.5 mm. from the caruncle. Simultaneously with this oblique movement the eyeball recedes 3 mm. into the orbit, and the palpebral fissure narrows to 4.5 mm.

When the gaze is directed up and to the right, the left eye shoots straight up. On looking directly upward, the left eye is directed obliquely up and to the left (Fig. 3). On looking up and to the left, the excursion of the left eye equals that of the right. In passing from the position of dextroversion (Fig. 2) to levoversion (Fig. 4), the ocular aspect changes in the following respects: the right eye makes a full excursion to the left; the left eye moves obliquely down and to the left until it reaches a position about 20 degrees beyond the median line, where it comes to a full stop. In addition, the globe comes forward until it reaches the plane of the right globe. At the same time the right palpebral fissure narrows to 6 mm. and the left fissure widens to 8 mm.

The attempt to look directly down is represented on the left side by an oblique down and out movement of the left eye. When the gaze is directed down and to the right, the left eye follows the movement of the right. Movement down and to the left is well accomplished, the left eye passing through an arc of rotation equal to that of the right.

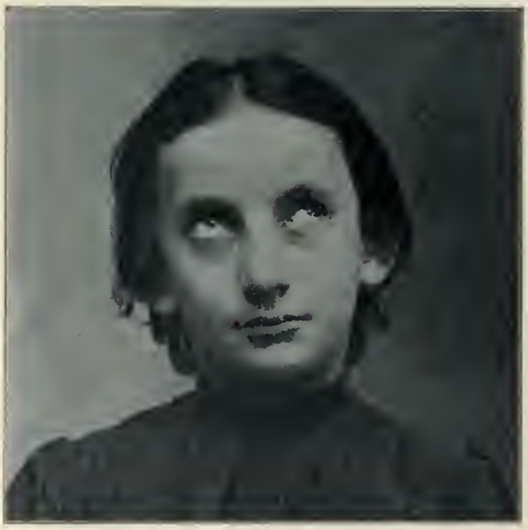

Figure 3.

If convergence be attempted, the right eye turns well in toward the nose, while the left remains motionless until the object is brought within 10 cm., when it diverges. If the near object be placed below the horizontal plane the motion of the left eye is straight down. With the right eye screened, the rotations of the left differ in no respect from its rotations in company with its fellow. After cocainization the tendon of the left internus was firmly grasped by fixation forceps and the globe easily rotated outward until the outer angle overlapped the corneal limbus. A similar attempt to rotate the globe inward by grasping the tendon of the externus was only partially successful, as the patient complained of pain and tension when the inner limbus was 3 mm. from the caruncle. The sensation imparted to the fingers holding the forceps was that of a definite resistance to further inward rotation.

The patient, then, presents all the features described by Duane as typical of the syndrome, i. e., partial absence of outward movement, partial deficiency of inward movement, retraction of the globe in adduction, a sharply oblique movement up and in and down and in in adduction, and, finally, paresis of convergence.

A search through the literature subsequent to the publication of Duane's paper yields two and possibly three additional examples of this anomaly. Jackson[2] reports the following case:

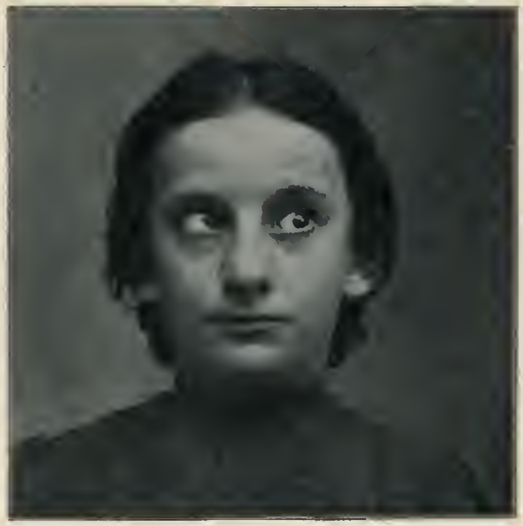

Figure 4.

In a 10-year-old boy with occasional slight internal squint, the right eye could not be turned to the right beyond the median line; the left eye could be turned to the left only 5 degrees beyond the median line. Movements in other directions were only slightly limited. On attempting to look to the right, the right eye came to the median line and opened widely; the left eye turned in, accompanied by narrowing of its palpebral fissure, and the eyeball was retracted 2 mm. or more. On attempting to look to the left, the right turned in and was retracted 2 mm. or more, while the left came to the median line and opened widely.

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[2] Ophthalmic Record, 1905, p. 248.

The following is a synopsis of a case reported by Harman[3] under the title, "(Functional) Absence of Internal and External Recti Muscles":

A girl, aged 16, presents no anomalies in the right eye. L. eye, V. 6/60, divergent 5 degrees. On directing the gaze to the right, the left eye moves in 20 degrees and is strongly retracted. Concomitantly, the lids "fall in" on the sunken globe, thus narrowing the palpebral fissure. In this position the lower lid is separated from the globe by a deep sulcus from 1 to 2 mm. broad. Attempted movement up and to the right evokes an oblique movement up and in of the left eye, accompanied by narrowing of the palpebral fissure. Attempted movement down and to the right evokes an oblique movement down and to the right with retraction of the globe and the formation of a sulcus between the lower lid and globe. In the effort to look to the left, the eye remains stationary, executing at times slight nystagmoid movements up and to the left and down and to the left.

The case reported by Raia,[4] under the title, "Congenital Paralysis of the Abducens of One Eye with Convergent Squint of the Other," while not indubitably belonging to this group, presents certain features of similarity:

A girl of 16 presented a slight convergence of the left eye. The right converges 10 mm. under cover, convergence persisting after removal of the screen. A distant object can be viewed with the left eye, but fixation soon passes to the right. When the gaze is directed to the left, the left eye fails to move past the median line, but the right eye makes a full excursion to the left, accompanied by narrowing of the palpebral fissure. There was no diplopia, the patient fixing with either eye at will.

Various theories have been advanced to account for the observed phenomena. As Duane remarks, it is inconceivable that the causative factors should be other than peripheral. The absence of abduction is explained on the supposition that the external rectus is absent or replaced by an elastic or inelastic connective tissue cord, a theory which in some cases has been borne out by anatomical findings. Certain authors, notably Heuck[5] and Harman,[3] ascribe the limitation of outward movement to a faulty insertion of the externus far back on the globe. Harman supports his explanation by comparative anatomy, having found that many of the salmon tribe and all of the herrings have the internal rectus inserted into the globe close beside the optic nerve, and that the externus is always faulty in growth and position in flounders. In the present case the definite resistance to passive traction inward (with forceps) seems to indicate that the externus is replaced by an inextensible cord.

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[3] Trans. Oph. Soc. United Kingdom, 1905, p. 281.

[4] Annals of Ophthalmology, July, 1905.

[5] Zehender's Klin. Monatsbl. f. Augenheil., 1879, p. 275.

The restriction of adduction is capable of two explanations: (1) With an inelastic externus, the normally inserted internus is thereby prevented from exercising its full function, or (2) the internus is inserted too far back on the globe to exert any notable inward rotation, this function being assumed somewhat inefficiently by the conjoined action of the superior and inferior recti. It seems probable that both explanations hold good for different cases.

If we assume that the externus is replaced by an inextensible cord, it is easy to see how the eyeball, being hugged between this cord on one side and the contracting internus on the other, must necessarily recede into the orbit. Some authors, however, agree with Harman that the "conjoined action of the superior and inferior recti muscles, by their oblique line of pull from origin to insertion, produce retraction of the globe." Duane opposes this theory on the ground that the obliques which would oppose this retraction must be assumed to be relaxed, which appears to be the opposite of the actual condition. According to Duane, the oblique movements observed in adduction "are due to spasmodic action of the inferior or superior oblique, probably often combined with spasm of the superior or inferior rectus." Harman, however, ascribes these movements to the "diverting of the eye up and down, when the object is fixed above or below the horizontal plane of action of the superior and inferior recti."

Much uncertainty exists as to the cause of the narrowing of the palpebral fissure when the affected eye is directed inward. It has been suggested by Parker that this is an example of a peculiar associated movement produced by synergic action of the facial and third nerve. This explanation can not be accepted for such cases as Harman's, in which the absence of any active contraction of the orbicularis is shown by the presence of a sulcus between the globe and lower lid. This author ascribes the narrowing of the palpebral fissure simply to a passive "falling in" of the lids on account of the lack of their natural support on the retraction of the globe. I am inclined to accept this explanation for my own case, as the lower lid, although in contact with the globe, was certainly not in a state of contraction.

The insufficiency of convergence appears to be due, as Duane suggests, "to the mechanical hampering of the action of the affected internus."

Beyond correcting the refractive error, little can be done in the way of treatment. In the few cases in which operation has been tried, the results have hardly justified the procedure. While tenotomy may be indicated in certain cases complicated by internal deviation, advancement is always contraindicated, "because it would increase the tension and hence the retraction" (Duane).

DISCUSSION.

Dr. Jackson, Denver, Colo.:—A striking clinical feature of these cases is the complete absence of diplopia in the affected part of the field, with the inability to get binocular vision. In the portion of the field where the patient can get binocular vision there is the ordinary response to diplopia tests, but the patient never notices diplopia. With reference to the pathology of my case, it seemed to me the retraction was due to the rotation of the globe, not around the ordinary center of rotation, but around a center somewhere in the region of the attachment of the defective externus muscle. The eye turned in around a center somewhere at the outer part of the orbit, and therefore moved backward as a whole, but not from any pressure of the lids.

CAN A SUBSTITUTE FOR SIMPLE ENUCLEATION BE EMPLOYED IN EVERY INSTANCE?

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The object of this paper is not so much to critically discuss the various methods which can be employed as a substitute for a simple enucleation as it is to call attention to the very pertinent fact that whenever an enucleation is indicated or performed some substitute which will yield a more prominent stump is not only indicated, but absolutely justifiable. Unless the surgeon does make an effort or employ a method to produce a prominent stump so as to minimize the future deformities, he is not doing full justice to his patient. Every operation about the face has two distinct and separate end-views; primarily and essentially, curative; secondarily, a cosmetic value. To operate only for cure or relief, unmindful of the ensuing deformity, is likewise not doing one's full duty towards his patient.

In the face of perhaps severe criticism, I do maintain, upon broad principles, that a substitute of the Frost type should and can be safely employed, either directly at the time of enucleation or at a near subsequent time. When it is deemed advisable to do the implantation subsequently, it should be done before firm cicatrization of the orbital tissues has taken place. By a subsequent time I mean generally within two or three weeks after the primary enucleation. Doing it later than this, though the cosmetic result is enhanced, the range of motion is not materially benefited because of the previous atrophy and misplaced muscles. It is as Grimsdale and Brewerton (page 155) state: "But if we are by operation to avert the danger of loss of both eyes, we must surely do our best to endeavor to minimize the resulting deformity, and hence, despite its simplicity, *simple removal is slowly but steadily losing ground, as a routine operation in all cases, while one of the other substitutes is adopted*." (Italics mine.) Only two methods are deemed worthy of serious consideration—the Mules and the Adam Frost type. All others yield comparatively insignificant prominency of the stump. Such methods as heteroplasty —either the implantation of animal eyes or tissues taken elsewhere from the body—do not yield sufficient uniform results as to warrant their application or even encouragement. All simple suture stumps, though better than no attempt at all, need no consideration, for I believe that every one who does an enucleation ought to employ the purse-string suture and secure at least a moderate lessening of the cavity. These suture stumps are easy of performance and do not prolong healing, and certainly are more surgical, as all wounds ought to be closed as rapidly as possible to allay suppuration and enhance primary union of the coaptated conjunctiva.

Of the two methods up for discussion, the writer is an ardent follower of the Frost type and not of the original or modified Mules operation. Of the materials employed for implantation only two are worthy of consideration, namely, lead-free glass and decalcified bone (Suker and Faith method), and, of these, lead-free glass is the choice. For any other material than this is, in the course of time, affected by the body juices, and we know that our economy furnishes no acids or alkalies that affect lead-free glass. It is needless to enter into this question any farther. We are all familiar with the literature and reports of cases dealing with this particular point.

It is a patent fact that capsule implantations can be employed in every case, whereas any other method of securing a prominent stump by implantation is only indicated in selected cases. One has a limited range of application, the other not. Therefore, why not employ it? No other substitute for simple enucleation than that of scleral or capsule implantation would yield as permanent and satisfactory a stump. The consistent employment of substitutes for enucleation by the writer for the past fifteen years has forced him to make this sweeping statement in favor of capsule implantation. No valid proofs have been presented which can substantially controvert this experience. Age itself is no contraindication for an implantation. In fact, it is even more obligatory in the young because it favors the uniform development of the socket. In young children about the age of 5 years or younger, if an eye is enucleated, that socket and side of the face is retarded in its growth unless a prominent stump over which a well-fitted shell can move is secured. Clinical experience has shown that a large movable stump as well as a full socket is essential for the uniform growth of a socket.

There is no real or valid surgical objection to simple enucleation. It possesses many advantages and all the disadvantages; the latter are the after-conditions, and these we ought not to forget, but make serious endeavors to obviate. These objections are:

First.—The orbital fat and muscles—even if a purse-string suture is used—atrophy and sink deeply into the orbit, leaving an extremely large cavity.

Second.—The upper lid invariably falls back into the orbit, leaving an unsightly hollow above. The lower only too frequently droops.

Third.—The stump movements are very imperfect.

Fourth.—The movement transmitted to the shell from this atrophied and shrunken stump is extremely and relatively small.

Fifth.—The vacant stare which is such a deformity is always present, so much so that many patients prefer an empty socket.

Sixth.—There is so much space between the concave shell and stump for the accumulation of secretions which frequently decompose and violently irritate the conjunctiva, causing the proverbial chronic conjunctivitis, and because of this discharge the shell easily becomes roughened, a point never to be disregarded.

Seventh.—Because of the above perpetual irritation, contracting adhesive bands are readily formed between Tenon's capsule and the subconjunctival tissue. Therefore, the cul-de-sac is gradually lessened in depth until finally it is so nearly obliterated that it is difficult to even insert a shell, to say nothing about wearing it constantly.

It is because of these seven very valid objections that I have for years urged the abandonment of the simple enucleation and have constantly and consistently practiced the implantation method. These implantations, however, to be only into the capsule of Tenon. For, though the Mules operation yields perhaps the most satisfactory cosmetic result, yet the globe is only too frequently extruded. Extruded, because the cornea, which is frequently retained, not being properly protected, gives way under friction, as all non-vascular tissues, particularly cicatricial, are liable to—as in reality the cornea becomes after an evisceration and a scleral implantation. The cosmetic value of Mules' as a whole is not any better or motion greater than in a properly performed implantation into the capsule cavity. My experience is not at all favorable to the Mules because of the extrusion, greater reaction and the possible danger that may attend the non-severance of the optic nerve. Unless a neurectomy accompany the Mules, the implanted ball often lies in close contact with the nerve, and may be the source of annoyance or reactions not unlike that of sympathetic irritation and inflammation. Then, too, it is rather difficult to get a perfectly fitting sphere into the scleral cavity, thus allowing a space between the sclera and the sphere. Furthermore, the sclera does not exhibit a great tendency for the formation of granulation tissue and, therefore, no such cushion surrounds the sphere as it does when the same is implanted into the capsule. This cushion is absolutely necessary for the permanent retention of the implanted sphere. In order not to have the sphere extruded so frequently in the Mules, it is best to amputate the cornea, freely dissect the conjunctiva back—if necessary even tenotomize the four recti muscles—and after the sphere has been inserted into the sclera, the latter properly sutured, repress the whole into the conjunctival sac produced and bring a good layer of the latter over the sclera. The two sets of sutures are to be at right angles to one another. This mode of operating insures the longest retention of a sphere for the Mules method.

When an eye is in an active state of suppuration, an enucleation is perhaps attended with some dangers—principally that of perforation of the globe at the time of operation and the allowance of infection of the orbital tissues thereby. This may result, according to some case reports, in meningitis. It has been for this reason, and this reason alone, that methods have been tried which would fully answer as a substitute for enucleation, and these, in turn, have led up to the Frost and Mules methods of implanting a foreign body either into the capsule or scleral cavity.

If meningitis does follow an enucleation, it is not due to an extension of the inferior along the nerve sheaths, but to an infection through the orbital roof. This is so because of free drainage and the direction of the lymph current, which is from the skull forward along the optic sheaths and emissary veins.

The element of time requisite for the healing of a capsule implantation should not argue against its employment. It usually takes about ten days before the patient can resume his work, and within three weeks from the time of operation he can safely wear a shell. Though operators hold the element of time up against any other method than the simple enucleation, it is not a sufficient cause or reason not to perform implantations whenever possible. The only time when implantations can not be done immediately at the time of enucleation is, perhaps, when one is dealing with a suppurating globe. Then it may be advisable to defer implantation for a short time. Under such conditions one can eviscerate, wait until suppuration has ceased, then amputate the scleral cup, and proceed in a manner like unto the simple capsule implantation. In case a Mules' is unsuccessful or the sphere is subsequently extruded, the scleral cup can be enucleated and the self-same sphere implanted into the capsule cavity. It is never wise to again attempt a scleral implantation when once there has been an extrusion.

Not even intraocular tumors militate against capsule implantation. This certainly, however, with the proviso that the globe was enucleated before perforation has taken place, and that metastatic nodes have not appeared in the orbital tissue. The writer has two such cases, both sarcoma of the chorioid, in which he performed a capsule implantation some two years ago, and up to the present writing no evil consequences have supervened because of said implantations.

No major operation—and certainly an enucleation is a major one—is always attended with proverbial success. Failures are bound to occur. But what harm is there, even if after a lapse of time, the implanted sphere, for some cause or another, is extruded? You have not jeopardized the safety of the patient or the fellow eye in any way. For, as has been conclusively proved (de Schweinitz, Mules, Suker), these implantations into scleral or capsule cavity have not produced a clear-cut case of sympathetic ophthalmia, provided that they were performed in the proper manner and at such a time when simple enucleation itself would have prevented such disastrous complications in the fellow eye. These latter remarks hold true only for the Mules operation and need not be considered in case of simple capsule implantation. Certainly the implantations into capsule cavity are absolutely unable to produce sympathetic reactions, for obvious reasons, the eye having been removed. Should, however, sympathetic ophthalmia ensue, it would have ensued even if no implantation had been performed, as the seeds thereof had already gone beyond operative relief. This argument of sympathetic inflammation following capsule implantation is not founded upon good reasoning, and to a large measure the same holds true for the Mules'. An implantation into the scleral cup can only produce so-called sympathetic irritation and not inflammation, just as any stump may cause an irritation, and then the removal of the sclera followed by a capsule implantation will be a satisfactory and justifiable procedure. It will be found that in those cases where sympathetic irritation follows either scleral or capsule implantations the wearing of a shell itself is frequently a sufficient cause for the reaction. It is granted that sympathetic irritation does not follow capsule implantation as often as it does scleral implantation. This for the reason that none of the essential ocular tissues are retained in executing a capsule implantation. Before attributing the irritation to either method the patient should not be allowed to wear a prosthesis for some time in order to see whether or not the stump itself be the real cause or the two conditions combined—the stump and shell being the factors. If the former, it is a very simple matter to remove the whole stump or the implantation proper.

It is deemed essential, in either simple enucleation or its various substitutes, that the patient forcibly and regularly exercise the lids in order to prevent any sinking in of the upper or drooping of the lower. The act of forcibly winking is an admirable procedure for them to perform. So also is massage of the lids. It is also essential that the prosthesis fits accurately in order to allow perfect closure of the lids. Furthermore, the sinking and drooping of the upper and lower lid is further guarded against by preserving the function of the sympathetic nerve fibers which supply the two. For this reason do not open the capsule posteriorly any more than just sufficient to permit the passage of an instrument for the severing of the nerve. This sunken-in appearance and the drooping of the lids can in a measure be ascribed to the enophthalmic conditions; that is, the stump is comparable to a traumatic enophthalmos.

A word or two with reference to the operation. For the implantation the sphere is boiled for several hours in a 1-5000 bichlorid solution, then washed in a saline solution before inserted into the capsule cavity, after a very careful enucleation has been made, preserving every particle of tissue possible.

The muscles and some capsule tissue are now brought over the sphere by a catgut purse-string suture. After this, the conjunctival and subconjunctival tissue is drawn by a series of interrupted sutures over the muscles and sphere. As soon as possible an ice pack is applied to the part for several hours, to be repeated two or three times a day for two consecutive days. The ice application allays any marked reaction or edema. The conjunctival sutures are usually removed within a week or ten days, depending upon conditions.

DISCUSSION.

Dr. **Baker**, Cleveland, Ohio:—My own experience with all kinds of implantations has been unsatisfactory, so that I have gone back to the glass eye without implantation. I believe if we hope to get a good appearing result we must not injure the orbital tissues. I find the cases I have difficulty in getting a good appearance is where somebody else has made the enucleation. If you stick your knife or scissors through Tenon's capsule you will get bands of adhesion and the artificial eye will not move. If the enucleation is made properly and the artificial eye inserted two or three days after the enucleation, there will not be the sinking of the orbital tissues, the muscles do not lose their tonicity and they move the artificial eye as they did the original one. My results have been very good and have continued so for years.

Dr. **Fridenberg**, New York:—The method given has two objections: One, the including of a foreign body in the tissues of the eye, and the other the added danger of any injury to the eye which contains a glass globe under the conjunctiva. A blow which would not cause other trouble might fracture a glass globe. The glass globe sometimes proves a source of great irritation. As to the effect of the simple enucleation, I have not found bad cosmetic effects if we have the eye made to order. I think in addition to careful operation we have a great aid in the use of the so-called Snellen reformed eye and get good results. There are occasionally cases where you have a small stump, and the Snellen eye is not sufficient, and in these I have been using a small Mule's sphere inserted over the stump and under the artificial eye as a movable prosthesis. If the size is right there is absolutely no irritation and there is free movement. The advantage is that you can remove the globe and keep it out until it is possible to put it in. The ball lies loose in the conjunctival sac. (Dr. Fridenberg illustrated the device on the board.)

Dr. Alt:—Being a believer in the direct transmission of the inflammation from one eye to the other by way of the optic nerve and intervaginal space, I have always been satisfied with the simple enucleation carefully performed, and have never made use of substitutes. However, Dr. Fridenberg's, if I may call it so, "ball-bearing artificial shell" appeals to me as a good solution of this problem.

Dr. Green, Jr., St. Louis, Mo.:—I would ask Dr. Suker if he has had any experience with paraffin implantations, or the gold ball of Fox?

Dr. Suker (closing discussion):—In answer to Dr. Fridenberg's objection that foreign bodies should not be inserted into the human economy, he has but to compare the statistics of implantation and non-implantation. There is seldom, if ever, any irritation. Some of these implantations in my cases have been in for fifteen years without extrusion. As for the bursting of the hollow glass sphere, it may happen, but it does not expand; it falls in upon itself instead of forcing itself out into the tissues. They break like an electric light globe. The extra support for the shell, of which the Doctor has spoken, I have tried by using paraffin or plaster-of-Paris on the concave side of the shell, but the stump did shrink and the muscles did atrophy nevertheless, and the result was the same as in simple enucleation. You have to have a full movement as well as a prominent stump in order to insure against the non-sinking in of the upper lid. As Dr. Baker has remarked, it is the careless cutting into the orbital tissues which causes the simple enucleation to fail so often in its cosmetic value. The size of the socket should also be taken into consideration. In a small, sunken socket it would not make much difference, but in the case of a prominent globe it is essential to preserve the fulness of the socket. Do not lose sight of the motor effect of the sympathetic nerves on the upper and lower lids. The congenital absence of the sympathetic gives you ptosis, and the injury to the sympathetics in doing a careless enucleation often results in the sinking in of the upper lid, because of the ensuing traumatic sympathetic ptosis. I have used the paraffin, but, as I have stated in my paper, only lead-free glass is not affected by the animal juices, and while paraffin does not liquefy in the patient's socket, it is incorporated with a certain amount of vaselin and this is absorbed and the stump shrinks. The paraffin also shows a tendency to disintegrate. I can show you spheres that I have taken out which were exceedingly friable. As for gold spheres or gold nettings, they do corrode and will break down, thus necessitating a secondary operation. Certainly from my own experience and from animal experimentations which have covered quite a few cases as well as years, I emphatically state that we are not justified in disregarding implantations.

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Caplan, Leo...............................309 Century Bldg., St. Louis, Mo.
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Carpenter, J. T.......................1624 Walnut St., Philadelphia, Pa.
Carrow, Fleming..Detroit, Mich.
Case, George M...Elmira, N. Y.
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Chamberlain, J. W..........................Lowry Arcade, St. Paul, Minn
Chase, John ....Denver, Colo.
Cheatham, William....................303 W. Chestnut St., Louisville, Ky.
Cheney, F. E......................126 Commonwealth Ave., Boston, Mass.
Chittenden, E. N...........................211 E. 11th St., Anderson, Ind.

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Cline, Lewis	224 N. Meridian St., Indianapolis, Ind.
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von Colditz, G. T.	161 State St., Chicago, Ill.
Conboy, Phillip	109 East Ave., Rochester, N. Y.
Connor, Leartus	91 Lafayette Ave., Detroit, Mich.
Connor, Ray	91 Lafayette Ave., Detroit, Mich.
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Cott, George F.	85 N. Pearl St., Buffalo, N. Y.
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Dean, Harry J.	122 Iowa Ave., Muscatine, Ia.
Dean, L. W.	Iowa City, Ia.
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Dowling, Oscar	1-3 Cooper Bldg., Shreveport, Ia.
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Dudley, W. H.	640 H. W. Hellman Bldg., Los Angeles, Cal.
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Dunn, Oscar B.	Ironton, Ohio

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Emerson, Linn	Orange, N. J.
Enfield, Charles	Jefferson, Ia.
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Esterly, D. E.	735 Kansas Ave., Topeka, Kas.
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Fisher, W. A.............................103 State St., Chicago, Ill.
Fiske, George F..........................100 State St., Chicago, Ill.
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Flagg, John D..........................384 Franklin St., Buffalo, N. Y.
Forsyth, Edgar A.......................322 Franklin St., Buffalo, N. Y.
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Frank, Mortimer..........................103 State St., Chicago, Ill.
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Friedmann, A. C. H................2 N. Arcade, Colorado Springs, Colo.
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Parker, Walter................912 Chamber of Commerce, Detroit, Mich.
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Peters, Walter H......................606 Columbia St., Lafayette, Ind.
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Phinney, Frank D......................22 W. 70th St., Cincinnati, Ohio
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Willis, E. A. . . . . .Indianapolis, Ind.
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